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SURGICAL DISEASES OF THE ABDOMEN

WITH SPECIAL REFERENCE TO DIAGNOSIS

DOUGLAS

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SURGICAL DISEASES

OF THE

ABDOMEN

WITH SPECIAL REFERENCE TO DIAGNOSIS

BY

RICHARD DOUGLAS, M.D.

FORMERLY PROFESSOR OF GYNECOLOGY AND ABDOMINAL SURGERY, MEDICAL DEPARTMENT, VANDERBILT UNIVERSITY, NASHVILLE; EX-PRESIDENT OF THE SOUTHERN SURGICAL AND GYNECOLOGICAL ASSOCIATION; FELLOW OF AMERICAN ASSOCIATION OF OBSTETRICIANS AND GYNECOLOGISTS; MEMBER OF THE BRITISH GYNECOLOGICAL ASSOCIATION, ETC.

Illustrated by 20 Full Page Plates

SECOND EDITION, REVISED AND ENLARGED

EDITED BY

RICHARD A. BARR, B.A., M.D.

PROFESSOR OF ABDOMINAL SURGERY, MEDICAL DEPARTMENT, VANDERBILT UNIVERSITY

LATE MAJOR AND SURGEON, FIRST TENNESSEE INFANTRY, U. S. VOLUNTEERS

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I Dedicate This Work

To My Mother

SARA CRAGWELL DOUGLAS

PREFACE TO SECOND EDITION.

The unexampled accuracy and thoroughness with which Dr. Douglas did his work anticipated the necessity, in this edition, for more than such few changes from the original as would be required by actual advances made in our knowledge of surgical diseases of the abdomen since 1903.

The desirability of preserving the text intact, as far as possible, since it bears very noticeably the impress of his characteristic style is sufficient reason for making only essential changes.

In making these changes I feel that my association with Dr. Douglas and my knowledge of his work have enabled me to follow closely his own ideas.

RICHARD A. BARR.

PREFACE.

As a teacher and practical worker in the special field of abdominal surgery for eighteen years, I have found it necessary to rely largely upon Journal literature for guidance. Reliable sources of information have not always been available and on this account much of my work has been done in a manner to me unsatisfactory. I therefore undertook a systematic study of those conditions within the abdomen which require surgical relief, relying upon my clinical experience to aid me in the choice and method of consideration of subjects, and the selection from general literature of the material which has proved to me of greatest value. The facts here presented are only such as have seemed to me of practical usefulness and as have borne the test of application.

My effort has been to elucidate the difficulties of diagnosis by a more thorough study of the causes and nature of these conditions, and make possible a preparatory knowledge which not only is essential to perfection in operative technic, but which will deter the conscientious operator from undertaking tasks beyond his skill.

The description of operative technic is lavishly and profusely given in numerous text-books, and its reproduction here has been deemed unnecessary and somewhat opposed to the purpose of this work. I have regarded it sufficient to indicate the proper surgical procedure and to discuss some of the open questions involving the operative treatment and after management of abdominal cases.

I desire to express to Dr. W. M. L. Coplin my grateful appreciation of his interest and services in helpful suggestions and criticisms and of his oversight of the illustrations. It also affords me great pleasure to acknowledge the invaluable aid given me by Dr. John W. Hanner, now of the U. S. Army, throughout the preparation of the work. To Dr. George J. Lochboehler, of Washington, D. C., I am indebted for valuable research, abstracts, and translations. I am greatly indebted to Miss Irene Nisbit for faithful and efficient aid throughout the preparation of this work.

RICHARD DOUGLAS.

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SURGICAL DISEASES OF THE ABDOMEN

PERITONITIS.

DEFINITION.—An inflammation of the peritoneum excited by bacteria under inviting conditions.

The pathological and clinical similarity of all forms of peritonitis not of tubercular origin and the distinctive pathology and siemiology of tubercular peritonitis make a division into Tubercular and Non-tubercular inflammation essential for the purpose of study. Peritonitis of every variety not caused by the bacillus tuberculosis is essentially an acute process, though isolated cases of chronic course throughout have been described, and some of the sequels of the acute process are designated as chronic peritonitis.

Acute Peritonitis (Non-Tubercular):

ETIOLOGY.—The hypothetical structures of chemical and idiopathic peritonitis are builded upon sandy foundation, fast disappearing before the advancing tide of searching bacteriology. The assumption that peritonitis is an infectious process, if not wholly true, is practically so. It is the only working basis which assigns a definite etiology, explains the phenomena, and directs prophylaxis. To comprehend the nature of peritonitis one must appreciate that the local inflammation, with its more or less flagrant phenomena, is but one feature of the disease; the most serious and subtle element is the condition of toxemia. The former denotes the resistance encountered by microörganisms, the latter indicates the rapidity and degree of systemic absorption. Bacteriological studies of peritonitis demonstrate that infections of this membrane are attributable to the same pathogenic germs that are found in similar processes in other parts, even in other serous structures; yet the effects, when the peritoneum is involved, are very different, in explanation of which we find it necessary to review the studies that have been made upon the histology and physiology of the peritoneum, and the effects of bacterial invasion of it.

The peritoneum presents an extensive surface, possibly as great as the whole integument of the body, capable of absorbing in one hour from 3 per cent. to 8 per cent. of the entire body-weight. It consists of an endothelial covering upon a limiting membrane, beneath which there is a framework of connective tissue richly supplied with lymph-channels and blood-vessels. Muscatello's studies show that the endothelial cells rest in close apposition to each other, and that there are no stomata in any part of the peritoneal surface; but that beneath the endothelia of the diaphragmatic peritoneum, especially its central portion, there are groups of open lymph-spaces. He states positively that none of these spaces exist in the peritoneum covering any other part of the parietes or viscera (Clark). Muscatello's experiments further prove that absorption from the peritoneum is by the lymphatics, the blood-vessels playing an indifferent, if any, part in that action. The lymphatics of the diaphragmatic peritoneum alone are capable of receiving foreign granular bodies. Leucocytes are largely the carriers of foreign bodies from the peritoneum into the mediastinal lymph-glands, and thence to the general circulation. There is a normal force in the peritoneal cavity which carries fluids and foreign particles toward the diaphragm, regardless of the posture of the animal. Clark, who has made a most careful study of the absorptive power of the peritoneum, and who presents an epitome of Muscatello's researches, says that fluids and solids may pass through the endothelial layer of the peritoneum, the fluids in many places, and the solid particles only through the spaces in the diaphragm. Experimental and clinical observations abundantly prove the truth of Wegner's assertion that the normal peritoneum is capable of absorbing enormous quantities of fluids, and may dispose of comparatively large quantities of infectious matter. This absorptive function of the peritoneum is its protection against infection.

The following fact was established by Grawitz, that the peritoneum is capable of disposing of sterile fluids and solid bodies, and also of large numbers of saprophytic and pathogenic bacteria, provided that they are introduced without injury to the tissues themselves, and provided that there was not introduced with them, or quickly accumulating, a quantity of putrescible substances greater than the peritoneum could absorb or encapsulate

within one hour. The conditions that Grawitz held favorable for the growth and baneful effects of microörganisms were: presence of stagnant fluid, chemical or mechanical irritation of the peritoneum, and especially the presence of a peritoneal wound or abrasion. These observations are confirmed by Halsted, Barbacci, and Tavel and Lanz. Flexner says: "In order that pathogenic bacteria, introduced directly into the peritoneal cavity, may cause a peritonitis, general or circumscribed, evanescent or fatal, the normal condition of the peritoneum must in some way be modified." The introduction of pathogenic bacteria excites a reaction in the peritoneum; Wallgren has studied this especially in reference to streptococcus infection. Immediately after the intra-peritoneal inoculation an intense phagocytic action is started; later, the leucocytes laden with streptococci disappear, this disappearance being due to the destruction, or solution of the leucocytes and to their deportation by the lymphatics. The leucocytes antagonize the streptococci not only through their phagocytic action, but through their secretions, the alexins. Durham holds that the leucopenic stage is due to several factors: first, to the sticking or attachment of the leucocytes to the peritoneal membrane, especially the omentum; second, to their absorption by the lymph-channels; third, to their entanglement in clots; and fourth, to the actual destruction of cells. And he further holds that leucocytosis and the later appearance of macrophages are essential to recovery.

That phagocytosis is an essential part in the protection of the peritoneum all observers hold; but if the supply of fresh germs continues, or if colonies of the most vigorous and strong microorganisms continue to multiply, the leucocytes are unequal to combat them, and the hope against a spreading infection lies in the capacity of the peritoneum to absorb and dispose of the noxious elements. Any destruction or alteration of the endothelial surface of the peritoneum lessens the capacity for absorption and increases the danger of progressive infection. In mild infections phagocytosis is effective in destroying the germs; but when the infection is virulent only a few cells appear, and the rapid absorption of the poison may overwhelm the patient before the local reactionary phenomena occur. It is, therefore, apparent that the effects of peritoneal inoculation depend upon the number and

virulence of the infecting organisms, the presence of suitable conditions for growth, the degree of leucocytosis excited, and the capacity of the peritoneum to dispose of the microörganisms while they are yet limited in number.

The want of resistance of certain regions to infection, namely, the enteronic and diaphragmatic portions of the peritoneum, and the tolerance manifested by other parts, namely, the pelvic, pericecal, and pericolonic, are usually explained on the anatomical basis of the size and number of lymphatics. This is, in a measure, true, but the diffusion of the infection which takes place by peristalsis, so active in the enteronic area, is a factor that must be reckoned with.

Bacteriology: A bacteriological classification of peritonitis is beset with many difficulties and any effort to individualize the effect of a special bacterium, to connect its presence with definite pathological phenomena and characteristic symptoms, can be only tentative—the proof must rest upon bacteriological examination. That bacteria attack the peritoneum in different ways; that they possess a diversity of virulence and of action; and that a peritonitis excited by some varieties is more amenable to treatment than others, is proved; but we cannot by symptoms with certainty distinguish a streptococcus infection from one due to colon-bacillus. Mikulicz avers that all forms of peritonitis run the same clinical course regardless of the bacteria that cause them; but we are learning to distinguish clinically a streptococcus infection from one of less virulent organisms. There is a law laid down by Malvoz to the effect that if the peritonitis is of intestinal origin the colon-bacillus will show it; if it is of uterine origin—that is, connected with abortions or labor—we shall find the streptococcus.

Bacillus Coli Communis: It is asserted that the colon-bacillus constitutes nine-tenths of the bacteria of the digestive tract. Its behavior upon gaining entrance to the peritoneal cavity depends upon many circumstances, and, as Tavel and Lanz say, this bacillus has many morphological conditions, many forms and stages. The constancy of its presence, however, in peritonitis since the demonstrations of Welch, Ziegler, and others of its power of transmigration through the damaged or diseased bowel has led many to attach to it great significance as an etiological factor. Flexner and Brunn concede to the colon bacillus pyogenic

properties. In secondary, endogenous or visceral infection it is the predominating organism, but only occasionally is it found in pure culture; in forty-seven cases studied by Flexner it was present alone in nine and in combination in thirty-eight. According to Dudgeon and Sargent, it is the usual cause of Peritonitis complicating Appendicitis, and when present alone, that is without the Staphylococcus Albus, is very deadly, though less so than the Streptococcus and the Bacillus Pyocyaneus. The power of this bacterium to penetrate the tissues will explain its presence in the effusions, while the primary pathogenic microörganism is concealed in the involved tissues; as Curtis illustrates, it may be found in the pus of a peri-appendicular abscess in single culture, while the less vigorous streptococci are demonstrable only in the appendix. It is abundantly proved that simple impairment of the endothelial coat is all that is necessary to permit these microörganisms to penetrate the intestinal wall, and their growth and rapid multiplication is a natural consequence of the peritoneal inflammation. The finding of the bacillus coli communis in the peritoneal effusion, even in pure culture, does not necessarily prove that it was the primary exciting organism; and if it exists in mixed infection it has probably been engrafted upon and overgrown a less vigorous germ.

Streptococcus: In fatal suppurative peritonitis the demonstration of the colon-bacilli alone is accounted for by the abundant supply of these microörganisms and their vigorous growth, but even in such cases of endogenous infection there are frequently found streptococci in combination, and occasionally in pure culture. In acute diffuse peritonitis, and in all infections from without, the streptococcus is the most active agent. Bumm asserts that in streptococcus infection through the tubes and ovaries death may occur without macroscopical changes in the peritoneum. In 15 cases of puerperal peritonitis Fränkel found microorganisms, the majority being some variety of the streptococcus. Winckel, Bumm, Courtois-Suffit all hold with Fränkel that this microörganism is the most active agent in the production of puerperal peritonitis, especially those rapidly fatal forms. Fränkel demonstrates that the infection not infrequently occurs by way of the lymphatics, the microbes gaining entrance through genital lacerations and abrasions. Cases of streptococcus peritonitis following facial erysipelas are reported by Cheurlin, and by Rich-

ardson. Staphylococci usually appear in combination with the streptococci. Fränkel regards the streptococcus found post mortem in puerperal peritonitis identical with the streptococcus erysipelatis, and he succeeded in producing erysipelas by injection of pure cultures into the ear of a rabbit. In a recent case of general suppurative post-operative peritonitis facial erysipelas was a complication. In traumatic and post-operative peritonitis, and in those cases in which infection takes place from neighboring pus foci, the pyogenic organisms are usually streptococci, though such infections, as a general rule, are mixed.

Staphylococcus: The observations of Flexner give greater prominence to the staphylococci than has hitherto been ascribed to them. In 44 cases of exogenous infections resulting in secondary peritonitis, the bacterial cultures from the peritoneal fluid showed staphylococci present in 18 cases, in 14 as a mono-infection. The Staphylococcus Albus is present in almost every variety of Peritonitis according to Dudgeon and Sargent. When alone it is but mildly pathogenic. When peritoneal infection occurs gradually by extension through the wall of the bowel for instance, and not by perforation, the white staphylococcus usually appears first, and by producing extensive leucocytosis, influences favorably the prognosis of a colon bacillus infection following its wake. It is much less effective, however, when preceding an infection with the streptococcus or the bacillus pyocyaneus. The milky peritoneal exudate produced by the staphylococcus is loaded with active phagocytes and its removal at operation is of doubtful propriety.

The bacillus proteus, the bacillus aërogenes capsulatus, the bacillus pyocyaneus, and the bacillus typhosus are other infrequent causative microörganisms, generally found in mixed culture.

The pathogenic power of the gonococcus to produce a general peritonitis, though denied by Bumm, is now an established fact. The occurrence of a primary peritonitis incident to pneumococcus infection cannot be denied. Hence to the gonococcus of Neisser, and to the diplococcus pneumoniæ we must concede the property of producing a specific peritonitis.

Gonococcus: "Inasmuch as the gonococcus cannot survive in the peritoneal cavity it is doubtful whether peritonitis due to the gonococcus alone has any existence" (Treves). Discussing the action of streptococci upon the peritoneum, Senn says:

"Gonorrheal tubal pus cannot do this; its microbes do not find in the peritoneum conditions for their increase; the pus, therefore, acts as an aseptic foreign body." Statements from authorities of such eminence cannot be lightly ignored, and, indeed, have been universally accepted, notwithstanding the statement of Räther who, in 1890, asserted that gonorrhea was the most frequent cause of local peritonitis. Bumm, in 1889, limited the pathogenic property of the gonococcus to the superficial surface of the mucous membrane, claiming that it never enters the blood nor lymph-channels, and that it does not produce metrophlebitis nor general puerperal sepsis. Contrary to the observations of Bumm, in experiment Wertheim saw the bacteria of Neisser on the surface of the peritoneum creeping along under the serosa into the deep tissues and penetrating muscle-bundles. The period of their activity was not limited, however, until they had produced a sero-fibrinous peritonitis. He further obtained from pus-tubes pure cultures of gonococci. Menge, confirming the statements of Wertheim, made the additional observation that gonococci rarely form a symbiosis with other bacteria. These experiments and observations prove that the human peritoneum can be and is liable to inflammation by the gonococcus (Winckel). Following these observations, we find two cases of diffuse peritonitis from gonorrhea reported by Bröse. Then there appears the strong statement from that most accurate of observers, Sutton, that it is established beyond any doubt that this infection of the uterus may extend to the Fallopian tubes and, by extension, set up a peritonitis. The entire question is historically reviewed and carefully analyzed by Cushing. He reports two cases which he offers as the first convincing evidence of the existence of a diffuse general inflammation of the abdominal cavity caused by the gonococcus, and emphasizes the relation of the spread of the infection to menstruation, abortion, and parturition. Max von Brunn, discussing the entire question *pro* and *con*, says that peritonitis may occur in the male as well as in the female following the introduction of gonorrheal virus.

Undoubtedly then we do have gonococcal peritonitis and although this type of infection has a marked tendency to spontaneous localization through the plastic exudate that it causes, generalized peritonitis of purely gonorrheal origin is occasionally met with.

Pneumococcus (Micrococcus lanceolatus): Inflammation of the peritoneum, primary or secondary, consequent upon pneumococcus infection, is undoubtedly a rare disease; Netter found it in only two instances in 151 cases of pneumococcus infections. Cornil probably described the first case, in 1886. Since then the disease has been described by Ḍegos, Blackburn, Weichselbaum, Brunn, Flexner, Bryant, Cassaet, and others; one of the most carefully studied cases is that of Fisher, recently reported. It occurs at all ages, but in the young it is more common in girls; 11 of Brunn's 14 cases were in girls. According to Netter, the peritoneum is usually secondarily infected from the lungs, or from middle-ear disease. Brunn believes that primary infection in girls is chiefly through the genitals. Flexner reports two cases in which the infection occurred through the intestinal tract. Blackburn, in 1900, reports 38 cases of pneumococcus peritonitis. Pneumococcal peritonitis will usually manifest itself primarily as a generalized infection, but it has a strong tendency to later localize spontaneously and lead to abscess formation.

Other specific forms of peritonitis consequent upon diphtheria, scarlet fever, and actinomycosis, are not sufficiently worked out for classification.

Sufficient has been said to show that the bacteriological classification of peritonitis is as yet premature and unsubstantial, for the reason that the observations of the bacteriologist are either experimental, post-operative, or post-mortem, and therefore not generally applicable for clinical purposes. From out the confusion and uncertainty of a bacteriological study of peritonitis, we naturally seek for something more practical, and we accept as a beacon the admission of the bacteriologist that some other factor, mechanical or chemical, must be present to render the peritoneum vulnerable to the invading microörganisms. The disease supplying these conditions is the source, the initial lesion whence the bacteria gain their power; and such disease or condition is the parent of peritonitis. Peritonitis is but an extension of a primary pathological foyer; the only exceptions are those rare cases of hematogenetic infection, and those forms in which there is no ascertainable cause, and which honesty compels us to assign to the idiopathic group.

A classification, then, based upon the physical findings

appeals to us for its scientific exactness; it admits of clinical adaptation; it forever destroys the false hopes of medication and serum-therapy, and spurs the surgeon on to action, very definitely guiding him in his undertaking.

Various authors have classified peritonitis from an etiological standpoint. Grawitz, and Flexner distinguish a primary and a secondary peritonitis. Out of 864 cases Grawitz found only 13 of primary suppurative peritonitis, and in these there was noted a predisposing factor. Flexner characterizes peritonitis as primary when such exists without other organs being affected, the peritonitis resulting from metastasis by way of the blood or lymph from a distant focus of disease; and as secondary when peritonitis is the result of injury or disease of an organ of the abdominal cavity or of one in direct relation to it. The exogenous causations are synonymous with the infections from without of Mr. Treves; and the endogenous are due to infection from the viscera.

Peritonitis of demonstrable origin occurs from traumatic or consecutive causes; and as these terms are broadly used, perhaps it is well to explain that under the head of *traumatic peritonitis* are embraced all infections of this membrane arising from wounds and injuries of the abdomen or its viscera, whether accidental or operative, penetrating or non-penetrating. *Consecutive, secondary,* or *symptomatic peritonitis* may be subdivided into three varieties: *peritonitis by continuity, perforation-peritonitis,* and *metastatic peritonitis.*

I. **Traumatic Peritonitis:** We must embrace under this head all forms of peritonitis due primarily to a trauma. The injury or destruction of tissue, the retention of blood-clots, the escape of visceral contents, mechanically and chemically prepare a suitable soil for microbic invasion. The essential infecting element may be introduced from without, as through penetrating gunshot, stab, or operative wounds, under which circumstances it is usually a poly-infection, the streptococcus predominating. Contusions of the abdomen, without a parietal wound, lacerating or otherwise injuring viscera, and giving rise to intra-peritoneal hemorrhage, render the parts open to infection by the migration of germs from the intestines. A peritonitis so excited is sometimes spoken of as 'putrid'; while many varieties of microörgan-

isms may be present, the colon-bacillus is especially in evidence. Penetrating wounds involving hollow viscera may be said to give rise universally to peritonitis, the tendency of which is to become diffuse and to pursue an acute course. In contusions without rupture of hollow viscera the infection is gradually introduced, and its phenomena more circumscribed and subdued.

Peritonitis of post-operative origin, unlike other forms of traumatic peritonitis, occurs in individuals unable to supply resisting or limiting phagocytosis because of their general condition. Additional danger lies in the mechanical injury to the peritoneum and viscera. Consequently, an infection occurring under these conditions rapidly becomes general. It is true that modern asepticism has, to a great extent, eliminated this *bête noir* of the early laparotomist, yet its occurrence is sufficiently frequent with all of us to warrant emphasis upon the striking words of Nancrede: "No perfection of aseptic technic will invariably render innoxious faults or misfortunes of operative technic." The needless exposure of the peritoneum to chilled atmosphere, rude manipulations, the destruction of peritoneal epithelium by vigorous sponging, the failure to barricade with gauze the healthy tissues while dealing with an infected area, the neglect to cover all raw surfaces with peritoneum, the ligature *en masse* of great chunks of tissue, unnecessary, improper, or insufficient drainage, are some of the errors of technic which we commit in violation of the principles of surgery and common sense in our idolatrous attention to the almighty germ.

II. **Secondary** or **Consecutive Peritonitis**: The supervention of peritonitis secondary to preëxisting morbid conditions embraces in its etiology infective inflammations not only of all intraperitoneal viscera, but of all organs or tissues contiguous thereto; and it does not appear to me to be too broad if we include within its scope those peritonitides metastatic in nature, arising from a septic focus remote from the abdomen.

(*a*) *Peritonitis by continuity* is the uninterrupted extension of an inflammation from an infected area to the peritoneum; it is accomplished by microörganismal advance through the lymphatics, blood-channels, and by direct penetration of the tissues. The reactionary inflammation which determines localization or diffusion depends upon the virulence and number of the microörgan-

isms, the presence of stagnant or degenerating fluid in dead spaces, the degree of leucocytosis, the capacity of the peritoneum for absorption, and as Reichel, and McBurney emphasize, the individual resistance of the patient. Concerning this latter point, it is known that cardiac, renal, and malignant disease predispose to peritonitis. Puerperal peritonitis of streptococcus origin conspicuously illustrates inflammation by continuity. That there are local and constitutional conditions of the puerperal state rendering these patients susceptible to the diffusion of infection, which do not obtain under other circumstances, goes without saying. Yet septic areas in the liver, spleen, pancreas, kidneys, mesenteric glands, lungs, pleuræ, vertebræ, abdominal parietes, genito-urinary tract, which produce peritonitis by extension without perforation, are daily observations. Peritonitis of appendiceal origin is more frequent than that arising from any other condition. In extension by continuity the infection of the peritoneum occurs somewhat gradually, and the inflammation is thus limited in its extent, yet extensive infection of a virulent character, originating in the appendix, may give rise to a general and very acute peritoneal involvement. In all forms of infection by continuity the bacteriological examination generally shows streptococcus and colon-bacillus, though the bacillus proteus vulgaris has been found in pure culture (Flexner). In instances in which the appendix lies among the small intestines, though the infection may occur by continuity, the active peristalsis excited by vomiting and injudicious forcing of food causes its rapid diffusion. In peritonitis proceeding from incarcerated hernia Bönnecken finds in the fluid of the incarcerated hernial sac both cocci and bacilli; these germs have wandered out from the bowel even before gangrene takes place, and produce a fatal peritoneal sepsis, often without suppuration. It is the exception for a consecutive or secondary peritonitis to yield a pure culture; sooner or later the colon-bacillus plays its part, whatever that may be. In intestinal obstruction, with arrested peristalsis, stagnant circulation, noxious contents, we have the well-appointed stage, the *locus minoris resistentiæ*, upon which the bacillus coli plays the star engagement.

(*b*) *Perforation-Peritonitis:* When an ulcerative or gangrenous process establishes a direct communication between any of the

hollow viscera and the peritoneal cavity, a peritonitis so excited is a consecutive perforation-peritonitis, and the term should be limited to this special etiological origin; yet the sudden and unrestricted discharge of visceral or mural abscesses, leaking pyosalpinges, ruptured cysts, etc., are sometimes, I think, erroneously spoken of as causes of perforation-peritonitis. In perforation-peritonitis there escape in activity and virulence large numbers of bacteria, accompanied by the contents of the viscus. In this lesion we find all the essential requisites for destructive peritoneal infection: there is foreign material, often of greater amount than the peritoneum can absorb, chemical irritation, and microörganisms. It is not surprising, then, that the majority of cases of acute general peritonitis fall under the etiological class of perforation-peritonitis. The morbid process leading to the perforation may be exceedingly slow, as in latent duodenal or gastric ulceration; it may occur as the legitimate pathological result of typhoid or dysenteric ulceration, or with the storm of gangrenous appendicitis or acute phlegmonous cholecystitis. However that may be, the knowledge that perforation-peritonitis is a suppurative condition should direct us at once in our clinical investigation. And we know that certain areas of the peritoneum, certain districts of the abdominal cavity, the appendicular, cholecystic, enteronic, gastric, and duodenal, in the order named, are particularly liable to invasion by perforation; just as we know that the appendicular region and the pelvis are the most frequent sites of localized peritonitis from infection by continuity. The location of the perforation has other than a diagnostic bearing; unless the process has been gradual and nature has had time to establish adhesions, the more central and high in the abdominal cavity, the greater the tendency to diffusion, and more pronounced the symptoms of sepsis. I do not wish to convey the idea, however, that seems to possess more than one writer on this subject, that infection does not spread from the pelvis because it is uphill. Intra-peritoneal currents and intestinal peristalsis disseminate the infection. Force of gravity is a minor element in limiting peritonitis to the pelvis. Visceral perforation, whether traumatic or pathologic, is an ideal condition for germ-culture and the elaboration of toxines. Their rapid absorption and general diffusion sufficiently explain the grave state into which the patient is precipitated.

(*c*) *Metastatic Peritonitis:* Infectious processes remote from the abdomen may, by metastasis, give rise to peritonitis. Many of the forms of hematogenetic and cryptogenetic peritonitides are instances of transmission, either by the blood or lymphatics. Peritonitis of nephritic, rheumatic, pneumococcal, scarlatinal or erysipelatous origin is generally metastatic; rarely, if ever, primary.

PATHOLOGY.—The practical surgeon realizes that the reactionary inflammation visible upon the surface of the peritoneum incident to infection is a reflection of a hidden cause; and upon his ability to ascertain the seat and nature of this initial cause depend the thoroughness of his work and the ultimate issue of the case. All pathological reports that deal merely with the peritoneal changes are valueless. As elsewhere stated, these phenomena are oftentimes subordinated to the general condition of sepsis, which may be demonstrable only in the changes in the blood or in septic localizations in various parts. The pathology of peritonitis, therefore, comprises all causes which may in any possible manner supply an infecting agent to the peritoneum, and upon these points we may proceed with our investigations with a measurable degree of accurateness and definiteness. There yet remains for amplification the systemic state of sepsis, which, though often only symptomatically expressed, for anatomic and physiologic reasons peculiar to the peritoneum becomes the chief cause of death. We arrange for analytical study the pathology of peritonitis under three heads: (1) *Etiological;* (2) *Peritoneal;* (3) *Sequential.*

1. **Etiological Pathology:** It is unnecessary here to individualize the multitude of morbid processes which may act as foyers for peritonitis. These have been sufficiently considered in the general etiology. It is only necessary to emphasize that peritoneal infection has a source; and that the more familiar we are with the behavior of those primary infections or conditions out of which peritonitis may develop, the more alert shall we become to recognize its approach and progression.

The character of the peritonitis excited bears a very definite relation to the etiological condition. The site of entry alone influences the behavior independently of the peculiar microörganism; there is less resistance, mechanical, anatomical, and physio-

logical, to a central infection than to one entering other regions. The manner of entering is a very material factor; the sudden introduction of infectious material in large quantity, as occurs in visceral perforations, pathologic or traumatic, inaugurates phenomena widely different from the fortifying process observed in the peritoneum against an advance by continuity. The normal peritoneum antagonizes infection; the gravity of post-operative peritonitis is not due alone to the quantity and virulence of microörganisms, but to the impairment of the peritoneum by previous disease or rough usage. The capacity of the peritoneum to absorb sterile fluids, though extensive, has a limit; the retention of the excess invites infection from the intestines. The acuteness of the primary disease, which is largely due to the virulence of the microörganisms, transmits its fulminating type to the peritonitis incident thereto; a gangrenous appendicitis or cholecystitis begets a peritonitis essentially different in form from that surrounding a salpingitis or a malignant tumor.

2. **Peritoneal Pathology**: The peritoneal reaction excited by infection exhibits the phenomena of great cell-proliferation, leucocytosis, desquamation of endothelial cells, and a fluid exudation from blood-vessels and lymphatics. If Metchnikoff's views are correct the purpose of the phagocytes is to envelop, deport or destroy the microörganisms, or to sterilize the noxious principles, pending their removal by the lymphatics. These phagocytes are of two varieties, the micro- and the macro-phagocytes; the former correspond to the migrating leucocytes; the latter are large cells developed from fixed connective-tissue cells. Toupet observed very similar histological changes in what he styles 'aseptic' peritonitis in guinea-pigs, induced by injections of nitrate of silver. Durham, though adopting a different nomenclature, observed the same changes, accounting for the transient leucopenia by other factors than the absorption of the leucocytes, chiefly by their adherence to the omentum. The activity displayed at the site of infection has long been described as a mortal combat between the microörganisms and the leucocytes. This metaphor is retained, for it most aptly illustrates the phenomena. The leucocytes are assisted by the effusion of a fluid, the *liquor sanguinis;* the fibrinogen it contains comes in contact with the fibrin-ferment and para-globulin set free from the break-

ing-down leucocytes, and fibrin is formed confining the infection. When the microörganisms, though resisted, are in the ascendency, the barriers yield before them and progressive purulent peritonitis develops. The appearance of the peritoneum and the character of the exudate denote, on the one hand, the resistance of the tissues, and on the other, the destruction the microörganisms have wrought. From the macroscopical picture presented it is customary to classify peritonitis into various forms.

(*a*) *Serous Peritonitis:* Serous peritonitis is rarely met with as a pathological entity. It most usually appears in those mild infections associated with neoplasms. It is a frequent form of tuberculous infection. But there is a form, a chronic serous peritonitis, non-tuberculous in character. The serous exudate indicates that the infection is attributable to microörganisms of limited toxicity. It is, no doubt, of more frequent occurrence than observed, and is rapidly absorbed and disappears. Clinically it is generally found in association with the fibrino-plastic form, and may be local and encysted or general. When the fluid contains a free admixture of blood it is then spoken of as *sero-sanguineous;* this is denotive of a more severe infection. Encysted or diffuse serous effusions may surround or outline a more severe localization of the infection. The fluid, which is an albuminous exudate from the blood-vessels, contains a variable admixture of cellular elements, blood and fibrin, affecting its color and transparency.

(*b*) *Fibrino-Plastic Peritonitis:* In an infection of moderate severity the watery elements may be quickly absorbed or may, indeed, be limited in amount, yet a plastic exudate resulting in adhesions, constricting bands, and limiting walls of variable density appears. Adhesions are a sequel of infection and indicate nature's attempt at healing; and while in a sense protective, yet they are responsible for many disastrous complications in incomplete technic. The mildest irritation results in the formation of soft adhesions, which usually disappear; but if the irritation is continuous or chronic in character, material is constantly contributed to increase the strength and density of the adhesions. The newly-organized connective tissue or pseudo-membranes are regarded by Cohnheim as exudates, but as Virchow has shown, they are formed by changes in living matter. English and Ger-

man pathologists, and in this country Jones and Robinson, maintain that the endothelia are connective-tissue corpuscles, and that to the changes in these cells the construction of false membranes is due. If they maintain their connection with the cement-substance a formative inflammation results; while if the connection is broken between the inflammatory corpuscles they become pus-corpuscles (Heitzmann).

(*c*) *Fibrino-Purulent Peritonitis:* Incident to the continuous action of pyogenic microörganisms there is disintegration and liquefaction of the fibrous and cellular elements, and the fluid becomes turbid, brown, yellow, or greenish-yellow; is thin, watery, or of a creamy consistency; is often offensive and gaseous from admixture of visceral contents or presence of putrefactive bacteria. Such a purulent process may have advanced so slowly that it is completely circumscribed by a zone of fibrinous adhesions. The tendency of this localization is observed most frequently in the appendicular and pelvic regions, also met with in the form of a subdiaphragmatic abscess, and occasionally in the lesser omentum from pancreatic disease. In infections of greater virulence the purulent process advances rapidly throughout the peritoneum; the adhesions, though restraining, are insufficient to arrest its progress. Such a condition is described by Mikulicz as *progressive fibrino-purulent peritonitis,* more accurately descriptive of the true condition than the term general suppurative peritonitis. A suppurative peritonitis is rarely, if ever, general; some area of the peritoneum, walled off by adhesions, is uninvolved; yet at operation, unless the patient is eviscerated, the pus may appear to be generally distributed. A more usual picture of progressive purulent peritonitis is the deep congestion of the peritoneum, which has everywhere lost its luster; spots of ecchymosis may appear, and flakes of tenacious yellowish fibrin more or less cover its surface; the coils of intestine are lightly coherent, beneath and between which puddles of pus of varying consistency and color are found. The exudates in the various peritoneal pockets may be of different grades; here creamy pus, there a turbid or sanguineous serum, and more remotely clear serum may present, all ultimately to become purulent and putrescent.

(*d*) *Diffuse Septic Peritonitis:* This is always an acute process.

The term 'septic' refers to the general condition of the patient. Mr. Treves draws a sharp distinction in the varieties of peritonitis, and is unmistakably clear in his definition of this form, emphasizing its virulent toxic features and comparatively insignificant peritoneal changes. The German surgeons' conception of this variety of peritonitis is well expressed by Tietze, who defines it to be "that form of peritonitis in which there is little or no exudate, severe symptoms of intoxication, and terminating rapidly fatally." It is the "intestino-peritoneal septicemia" of Malcolm. It usually originates in perforation or post-operatively. The peritoneum is everywhere congested, frequently the effusion is limited to a few ounces of a brown ichorous fluid, the coils of intestine are distended and gluey; beyond this there may be no peritoneal phenomena. At the site of infection there are more marked changes, rarely of sufficient duration to result in any extensive suppuration. Bacterial cultures usually show streptococcus infection, though the colon-bacillus alone may be responsible.

(*e*) *Pseudo-Myxomatous Peritonitis:* A form of peritonitis characterized by a myxomatous growth or formation in the peritoneum, usually secondary to rupture of multilocular ovarian cysts and escape of gelatinous material. Ruder holds that the material does not grow upon the peritoneum, but accumulates in the peritoneum from the cyst.

3. **Sequential Pathology:** The peritoneum exhibits the so-called inflammatory features of peritonitis, but the changes here met with very poorly and insufficiently explain the condition of general sepsis. Peritonitis is lymphangitis (Robinson), and something more. The focus of inflammation supplies to the lymphatics fluids, corpuscles, and cellular elements laden with ptomains from bacterial action; this poison is first conducted to the mediastinal lymph-vessels and glands, thence to the general circulation for distribution. If the supply is limited in amount, no permanent ill effects follow. In copious and virulent infections the system is overwhelmed with the acute toxemia, and death results. It is only when the vital resistance is sufficient to withstand the shock of onset that we find marked peritoneal pathologic effects and secondary changes incident to disseminated sepsis.

It must be recognized that microörganisms do not abide upon the endothelium; there is penetration into the substance of viscera, and whether this infection is endogenous or exogenous, the involvement of the muscular wall of the alimentary canal inhibits its physiological function. Through nerve-irritation it becomes a paralyzed and passive tube, dilating under the retention of decomposing intestinal contents, from which are elaborated gases. Hence tympanites is a conspicuous pathologic and symptomatic feature of peritonitis, and thus, too, there is contributed an auto-intoxication of intestinal ptomains.

The early appearance of leucocytosis, from 18,000 to 40,000 per c.mm., shows the toxic state. The gradual increase in other cases indicates the progress of a local infection. In those forms of acute septic peritonitis terminating with the symptoms of shock, the patient cannot react against the poison and the leucocytic count may be normal or markedly below.

In ultra-acute cases the patient succumbs to paralysis of the vaso-motor and respiratory centers. In the sequential pathological progress of a peritonitis continuing over forty-eight hours, there may develop single or multiple localizations, such as pleurisy, pneumonia, endo- and pericarditis, meningitis, erysipelas, arthritis, or pyemia.

Symptoms.—Primary peritonitis is of such rare occurrence that we may eliminate it in the consideration of the general symptomatology. Regarding peritonitis for clinical purposes as a consecutive disease incident to some infecting focus, the anamnesis of the peritoneal pathological evolution involves the clinical phenomena of the initial disease. Manifestly, then, the appearance of peritonitis may be attended by the most obvious causes. Unfortunately, however, either from obscurity of symptoms, the latency of the primary pathological process, or the advanced state of the peritonitis, the etiological origin is exposed only by operative or post-mortem examination. The history of a perforation-peritonitis as a sequence to perforating typhoid ulcer is very different from the fulminating form consequent upon gangrenous appendicitis or cholecystitis. A knowledge of gonorrhea, with the development of peritonitis after abortion or menstruation, directs us to the seat of disease and its probable course. A history indicative of intestinal strangulation explains the phenom-

ena of the complicating peritonitis. When abdominal symptoms develop without assignable local cause, a knowledge of the occurrence of transmission-peritonitis may discover in the lungs, pleuræ, ears, or in a facial erysipelas the depot of distribution. The treatment of peritonitis is eminently operative, yet direct, precise, and successful surgery in a great measure depends upon our ability to reach the cause, which rests oftentimes upon the knowledge obtained through the history.

Mode of Onset: As we have found, there are many pathological expressions of peritonitis and just as variable are the modes of onset. The recognition accorded by the peritoneum to infection varies with the suddenness with which the infection occurs, the amount of infectious material, the virulence of the infection, the part of the peritoneum involved, the condition of the peritoneum, and the general condition of the patient. The actual development of symptoms of infection may be preceded by Abdominal Shock, or Peritonism, due to traumatism to the peritoneum or to the abdominal viscera. Peritonism is marked by severe abdominal pain, usually general over the abdomen, though it may be more intense over the area from which the infection came, or it may be referred chiefly to the Epigastrium or to the region of the Umbilicus. Vomiting, subnormal or at least a falling temperature, rapid weak pulse, cold clammy skin and anxious expression are noted. Local or general abdominal tenderness and rigidity of varying degree are found, depending upon the extent and degree of irritation of the parietal peritoneum. When the suddenness and the amount of irritation are not sufficient to cause shock or where the local or general sensorium is too benumbed to recognize traumatism, there is a development of symptoms varying in rapidity and degree with the factors already noted.

The symptoms of peritonitis are simply the local evidences of inflammation with the constitutional evidences of sepsis. Individual symptoms, local or constitutional, are so inconstant in their appearance that a better conception of the condition can be gotten by a discussion of the symptoms in detail rather than by attempting to draw a clinical picture. As we have seen, etiologically, bacteriologically and pathologically, we can recognize many varieties of peritonitis, but clinically we can recognize only localization and generalization of the process, and even here the dividing line is very inaccurately defined anatomically as well as clinically.

Despite the variable and inconstant nature of individual symptoms as described below, the clinical recognition of a developed peritonitis is not difficult, and, upon local rather than constitutional evidence, the degree of generalization can usually be judged with fair accuracy. The source of the infection can ordinarily be determined from the history of the case and upon local evidence, and from this and by the rapidity with which the process has spread we judge the probable infecting agent, a matter chiefly of prognostic importance. While the clinical recognition of a developed peritonitis is all too easy, the prompt recognition of a tendency on the part of an infection to spread from a local focus is next to impossible. In view of these facts, the clinical problem that confronts us in connection with peritonitis is the early recognition of conditions likely to lead to generalized peritoneal infection and the prompt institution of preventive measures.

Pain: Peritonitis may pursue its course to a *fatal termination without abdominal pain.* This, though an exceptional behavior, is observed in post-operative peritonitis, in non-tuberculous serous peritonitis in children, and in a few cases of progressive fibrino-purulent peritonitis. It is not implied that such patients are free from discomfort; the thoracic distress from distention may be very great. In perforation-peritonitis the onset of pain is very violent, and presents many phases; in one it may be sharp and lancinating or burning in character; it may be either continuous or paroxysmal with distinct remissions; it may be local or general. The sudden cessation of pain, in these cases especially, implies a gangrenous local condition with general diffusion, and is often indicative of that wide infection leading to the form known as *diffuse septic peritonitis,* which is quickly fatal. When the pain is distinctly colicky in character it implies some intestinal involvement with partial occlusion. This character of pain with its accompanying meteorism is characteristic of fibrinoplastic peritonitis. Acute localized peritonitis excites a lancinating, burning pain local to the point involved, or oftentimes referred. The site of pain does not point invariably to the seat of infection; in cholecystitis with peritonitis the pain may be referred to the appendiceal region, and the reverse may occur in appendiceal peritonitis; intestinal perforations, especially gunshot wounds, wherever located, may cause pain only in the um-

bilical region; but with the diffusion of infection the pain becomes general to the abdomen. In the acute onset general pain does not necessarily imply diffuse infection; nor is it to be inferred that local pain is an assurance that peritonitis is not general. An acute local pain subsiding, recurring subsequently as a general pain, is suggestive of grave trouble. Movement and change of posture usually exaggerate the pain in acute peritonitis. The condition of the alimentary canal influences it. In pelvic peritonitis defecation and urination are both painful. Menstruation and intercourse also produce exacerbations of pain in this form of peritonitis. Exercise usually excites pain in chronic peritonitis. The pain of peritonitis has longer duration, is more constant than that of hepatic, nephritic, or intestinal colic. Patients dying of peritonitis are insensible to the pain some hours before death.

Vomiting: The vomiting of peritonitis is a symptom originating in many causes. Severe abdominal pain alone may produce it; peritoneal irritation from foreign material excites it; it is often toxic in nature; the antiperistalsis excited by decomposing intestinal contents, which irritate the gastric and intestinal mucous membranes, is an additional cause of the vomiting in severe cases; the distention, especially gastric, due to duodenal constriction by the mesenteric vessels consequent upon enteroptosis, gives the regurgitant vomiting without nausea; in severe peritonitis with arrest of renal function, uremic poisoning produces vomiting and adds to the gravity of other symptoms. There are many factors which influence the symptom vomiting. It may be generally stated that there is a direct relation between distention, constipation, and vomiting; the more acute and painful the onset the earlier and more pronounced the vomiting; in central infections attended by grave nerve-disturbance and rapid absorption, vomiting is conspicuous; if bowels are active it is not usually pronounced; improper feeding will generally excite it; medication, especially opium, may control or modify it. In acute adhesive and in chronic fibrino-plastic peritonitis vomiting occurs only as a symptom of intestinal constriction. In acute localized peritonitis occasional vomiting usually attends the onset, subsiding when the stomach is empty, and when circumscription of the inflammation has been accomplished. A reoccurrence after an

interval of repose in such cases suggests extending infection. When vomiting and diarrhea are coexisting, usually without tympany, the vomiting is not so much the symptom of peritoneal irritation as it is of the state of general septicemia. In post-operative cases the gastric distention, followed in a few hours by vomiting, may be the earliest symptom of peritonitis. In puerperal peritonitis vomiting is a late symptom except in the fulminant types. In perforation-peritonitis, vomiting, like all other symptoms, is pronounced. In those cases of gastric perforation in which vomiting is inconspicuous, the perforation is small and the extravasation slight, as a rule. All cases of diffuse infection, of the violent septic type, are characterized by vomiting at first. Peritonitis originating in intestinal obstruction is, of course, usually preceded by vomiting, a symptom of the intestinal constriction; in such cases the vomiting often ceases with the full establishment of peritonitis.

The character of the vomitus in peritonitis is: first, the contents of the stomach; then a mucous, bile-stained pea-green fluid; and later, copious quantities of brown, offensive, sometimes stercoraceous matter. When the vomitus is black or chocolate-colored it is due to the admixture of blood and is a grave symptom.

The manner of vomiting is somewhat characteristic. Much nausea, heaviness, and epigastric distress may precede the occurrence of vomiting, as in post-operative cases; the free discharge of the accumulated stomach-contents is attended in such cases by much relief; belching occurs in the intervals; soon there is recurrence of the phenomena, the vomiting now becomes more frequent, a mere mechanical regurgitation of a mouthful of offensive fluid.

In peritonitis the rejection of all ingesta a few minutes after taking, and the continuous regurgitant vomiting are the characteristic features. When vomiting ceases, and the bowels remain persistently constipated, with increasing tympany, fast and failing pulse, the case is near its end.

Constipation: The suspension of the bowel-function with constipation of both gases and feces is an alarming symptom in peritonitis. It is difficult to explain this phenomenon. In acute general peritonitis the inhibition of the vermicular movement may be attributed to the inflammation of the muscular coat; yet

we find that this symptom, constipation, prevails for a time, at least, in acute localized peritonitis, and for want of better explanation is attributed to peripheral nerve-irritation. Bowel-stasis is more valuable than either pain or vomiting, as either or both of these may occur from a variety of causes; but this symptom of intestinal obstruction is found only in peritonitis, mechanical obstruction, and in the rare paralytic ileus. In peritonitis there is suspension of all muscular action, while in mechanical obstruction the converse is the case for a time at least. In virulent spreading infection, from whatever cause, constipation is, as a rule, very marked; a little flatus may be discharged under the persuasion of an enema; all efforts to secure free bowel-action are usually unsuccessful. This idea is so generally fixed in the minds of surgeons, that, in post-operative cases especially, a free bowel-movement is looked upon as an assurance that the prime danger has passed.

In the diffuse septic peritonitis the cardinal symptoms are the general state of the patient and constipation; the pain and vomiting may both be transient symptoms, but the symptom of obstruction is so absolute that these cases are often mistaken for mechanical ileus. In progressive fibrino-purulent peritonitis the obstruction may be mechanical as well as from other causes; this is especially true and frequently noted when the condition arises from appendiceal inflammation. Other symptoms, in particular pain, vomiting, and meteorism, are more or less dependent upon the state of intestinal torpidity.

While constipation is the prevailing condition in peritonitis diarrhea is the rule throughout the course of a pneumococcal infection and occasionally occurs in other types, especially in those subacute cases attended by the accumulation of large quantities of pus. (Empyema of the abdomen.) Diarrhea is not infrequent in puerperal peritonitis.

The symptoms thus far considered, pain, vomiting, and constipation, taken collectively, are suggestive of peritoneal involvement.

Pulse and Temperature: Some exception must be taken to what is styled the characteristic hard, snappy, whip-cord pulse of peritonitis. The pulse, like all other symptoms, is uncertain and variable. It may be rendered temporarily frequent from

several causes, as from vomiting, meteorism, etc. Its increasing frequency in progressive infection is a characteristic, if it has any. In perforation-peritonitis the pulse is soft, distant, and dicrotic. If diffuse septic peritonitis follows, the pulse may remain thready and weak, and we note hourly acceleration. Should reaction take place, the pulse seems to steady itself and remains slow for a time, before it takes on its snappy character and becomes rapid. In the onset of acute localized peritonitis the pulse may be very fast and hard, but when the infection is limited there is a marked decline in the frequency of the pulse. In such inflammations the pulse is especially valuable in denoting an accession of peritoneal infection. When a second rise takes place it indicates renewed microörganismal activity, and demands interference. A fast pulse for twenty-four or forty-eight hours with other symptoms means serious progress. In peritoneal infection a pulse of over 100 is of grave import. The pulse may be slow, in discord with other symptoms. I once saw a case of diffuse septic peritonitis in which the pulse twenty-four hours after the invasion beat only 65, while the temperature was only 99°, the patient dying in about fifty hours from the onset of symptoms. Localized abscesses or even progressive fibrino-purulent peritonitis may exist with a pulse of 90 and of good quality. All cases of fatal termination have in their ending a rapid and failing pulse.

The temperature is most misleading; it is often normal or subnormal, is rarely in accord with the general symptoms, and does not indicate the extent of the pathological process. Circumscribed peritoneal suppurations in their onset may be marked by high temperature, yet both pulse and temperature may steadily fall under dangerous, advancing suppuration. In diffuse septic peritonitis, if there has been an antecedent febrile condition, as in typhoid fever, the occurrence of the perforation and general infection are marked by a drop in the temperature. With recovery from shock the reactionary temperature in these cases of profound sepsis rarely runs high. An ante-mortem hyperpyrexia of 106° or 107° is not unusual. A review of many of the cases operated upon for diffuse septic peritonitis shows that the prevailing temperature is under 100°, and often below normal. In the less acute cases characterized by progressive fibrino-purulent pathological changes, marked variations in the temperature are

noted. In metastatic localized peritonitis a truly septic chart is obtained. Streptococcus infection, especially in puerperal conditions, shows at first slight but gradually rising temperature. In any case with abdominal symptoms showing high temperature for over forty-eight hours, septic infection may be inferred. If the temperature should suddenly fall, yet rapid pulse and local symptoms still continue, it means spreading infection with suppuration. While the exception rather than the rule we do at times have the onset of peritonitis with a chill, especially is this true when the process is of puerperal origin and in the perforated form rarely repeated distinct chills occur, though chilly sensations are more common.

Respiration: The breathing in acute peritonitis is characteristic; it is thoracic, shallow, and frequent. It is rendered so in the onset by the rigidity of the abdominal and thoracic muscles and the instinctive effort to keep the parts at rest to avoid pain. When the abdomen is distended by effusion and meteorism there is voluntary and often inflammatory fixation of the diaphragm, and compression of the thoracic viscera by the over-full abdomen. The restricted respiration and feeble heart-action are evidenced in cyanosis. Respiration is not affected in chronic peritonitis unless there is extensive effusion.

Insomnia: An uneasy wakefulness attends all acute forms of peritonitis.

In addition to the above somewhat positive symptoms there are indefinite and more variable ones to which attention must be called. In an acute onset of violent peritonitis there is shock, designated by Gübler as "peritonism," an expressive term embracing all those symptoms that arise from sudden peritoneal irritation. Mr. Treves very aptly likens the general condition of a patient with septic peritonitis to that of a man suffering from a snake-bite. The mental state of the patient is one of the most interesting and yet distressing features of peritonitis. When the infection becomes general, and before the end is reached, there often appears that dreaded state of euphoria; in the clear intellect, bright eye, and resonant voice the friends find false encouragement. Fortunately, such harrowing death-scenes are often spared us; after some hours of ceaseless vomiting and merciless thirst the patient passes into a stupor, in which state he dies.

The *facies abdominalis* is a late symptom and does not always appear. There is, however, about the patient a general expression of serious illness.

PHYSICAL SIGNS.—**Posture:** At first the patient lies quiet with limbs flexed and prefers the shoulders elevated. In certain forms of infection he may prefer a slightly lateral decubitus. His position is one of enforced rest and protection. Later, with the appearance of the general symptoms, he is restless, his arms are in constant motion, his body sinks deeper in the bed. With abdominal distention and compressed lungs and heart, he insists upon greater elevation of the shoulders.

Appearance of Abdominal Surface: In intensely septic cases the abdomen appears cyanotic and mottled. When there is great distention the skin is pale and glistening. In suppurative conditions the appearance of vesicles and pustules upon the abdomen presages a hopeless condition; I have found this in connection with erysipelatous infection. Localized suppurative peritonitis is frequently marked by edema of the parietes. The shape of the abdomen depends upon the cause and degree of distention.

Tenderness: Tenderness is one of the most constant signs in peritonitis. It is especially valuable in determining the seat of infection; McBurney's point in appendicitis, tip of the tenth rib in cholecystitis, and the left epigastric triangle in gastric perforation, are all very valuable clinically. In acute general infections there is hyperesthesia over the entire abdomen, which may in a few hours become localized at the seat of primary involvement. When local tenderness suddenly becomes general we should apprehend general peritonitis. In subacute conditions attended by extensive suppuration, the tenderness is sometimes surprisingly slight. The sudden disappearance of tenderness with persistency of other abdominal symptoms is an ill omen, indicating great toxicity. When tenderness is detected by digital examination through the anterior rectal wall as high up as the finger can reach, it points to either general peritonitis or a pelvic position of an inflamed appendix; in women, of course, one must exclude diseases of the appendages. Palpation in the same way may detect effusion, which is further significant of peritonitis.

Muscular Rigidity: There are two distinct types of rigidity of the abdominal muscles resulting from inflammatory troubles be-

neath. When there is no involvment of the parietal peritoneum the rigidity is distinctly voluntary due to the effort on the part of the patient to protect the tender viscera from manipulation. With involvement of the parietal peritoneum we have the development of an involuntary board-like rigidity that is present at all times and never relaxes until overcome by the persistent pressure due to the tension of gas in the bowels. When the peritoneum has been prepared, so to speak, for the invasion, the muscular reflex is not so great. And in those individuals whose vitality is greatly impaired by previous illness, it is also less marked. Allowing for these infrequent exceptions, muscular rigidity remains one of the most important physical signs of acute peritoneal infection, and naturally is especially conspicuous in perforation-peritonitis. In general infections that pursue a subacute course rigidity may not appear. Rigidity is not present in post-operative peritonitis. The value of this sign in diagnosis is its early appearance; if the case is seen after twelve hours, and sometimes earlier, the rigidity is displaced by distention.

Abdominal Distention: Whether it follows rigidity or occurs independently, the abdominal distention is attributable to dilatation of and accumulation of fluid in the stomach, to the effusion and putrefaction of fluid free in the peritoneal cavity, and to meteorism. Distention is generally symmetrical, giving the abdomen a uniformly arched appearance, pushing up the diaphragm, and displacing the thoracic viscera. Much of the abdominal distention is due to accumulating fluids in the stomach; this assertion of Mr. Treves I fully endorse, especially as applicable in the first stage of post-operative peritonitis. Gastric distention occurs usually as an early sign; so long as the distention can be relieved by lavage the prognosis is not altogether unfavorable. Peritoneal effusion may sometimes be detected before the advent of intestinal distention; if so, it conclusively differentiates peritonitis from intestinal obstruction, for in strangulation meteorism precedes the effusion. In very fulminant cases of peritonitis there is only moderate effusion, and general distention of both small and large intestines rapidly occurs. It is these cases that so closely resemble intestinal obstruction; and if we can determine a flaccid state of the lower bowel it points to obstruction and not to peritonitis. The mechanism of meteorism is not fully

explained; it occurs whether the intestines be full or empty, and while greatly increased by decomposing intestinal contents, the gas is a bacterial product, which may be generated in a very limited medium. It was the teaching of Mr. Tait that meteorism was a necessary accompaniment of all fatal cases of peritonitis. Experience scarcely sustains this; in those subacute peritoneal suppurations attended by diarrhea, meteorism may be absent throughout. The pressure-effects of abdominal distention, through the embarrassment inflicted upon the thoracic viscera, contribute to the patient's rapid decline.

Abnormal Areas of Resonance: The development of abnormal areas of resonance, especially over the splenic and hepatic regions, still appears in text-books as a constant sign of perforation-peritonitis. The inconstancy of the sign when perforation has been proved to exist, the many chances of error due to displacement of distended intestinal coils, and the fact that gas may be evolved in the peritoneal cavity without perforation, negative its value.

Tumor: A localized tumor most frequently occurs secondarily to visceral disease of a suppurative form; appendicitis, salpingitis, hepatic abscess are instances. The tumor-mass is composed of the thickened and inflamed viscus, of adherent intestines and omentum, plastic exudate, and contained within all pus. In general peritonitis running several weeks, especially of the fibrino-purulent type, isolated swellings may be encountered, due to the inclosure of serous or purulent matter by adhesions. In chronic peritonitis other than tuberculous, the thickening and rolling of the omentum and adhesions may form ill-defined areas of induration. The inflammatory tumors, the cause or the result of peritonitis, may vary greatly in their apparent size with the accumulation and disappearance of intestinal gases. They are usually very sensitive under pressure, often resonant upon light percussion, may be elastic or fluctuant.

PROGNOSIS.—The rapidity with which toxines are absorbed determines the condition of sepsis, and upon this the prognosis in any case of peritonitis largely depends. The dissemination of infection favors toxemia, and therefore the more pronounced the local reaction to the infection the greater the probability of adhesive and circumscribing inflammation. This evidence of nature's resistance affords occasion and assistance to the surgeon.

Those peritonitides developing in the regions of the gall-bladder, appendix, and Fallopian tubes are usually favorable instances for localization, unless the causal disease is ulcerative or gangrenous in character.

Perforation-peritonitis, wherever originating, so overwhelms the peritoneum with microörganisms and is attended by such favorable conditions for culture, that the usual and expected behavior is general diffusion with pronounced systemic poisoning; yet even in peritonitis arising from this cause clinical experience teaches that when the appendix is the seat of perforation or gangrene the resulting peritonitis, local or general, is more amenable to treatment than that from intestinal perforation. An acute peritonitis originating from virulent infection of the gall-bladder may rest within limitations for a short time, but the constant supply of infected bile precludes any hope of permanent localization.

When general peritonitis of the progressive fibrino-purulent character develops somewhat gradually, the prognosis is not altogether bad; a fatal prognosis based upon the quantity of pus and its apparent general distribution is often proved erroneous. In such cases the ultimate issue largely depends upon our ability to reach the cause and upon the choice of the proper technic in caring for and disposing of the effects of the inflammation.

If we restrict *diffuse septic peritonitis* to the class of cases designated in this article, medicinally treated they are all fatal; a sweeping statement which does not apply to the milder grades of inflammation apparently general yet attended by pus-formation. Nature may, by spontaneous evacuation, rescue the patient; such a forlorn hope, though, is better forgotten than remembered.

Cushing thinks that gonorrheal peritonitis with extensive infection may yield to medical measures. Pneumococcus peritonitis is not so rapidly fatal as other forms attended by suppuration. Of 14 cases of pneumococcus peritonitis collected by Brun 11 recovered, one, without intervention, by spontaneous discharge at the umbilicus; Cassaet places the operative mortality at about 20 per cent.

Post-operative peritonitis developing within a few days is singularly fatal.

The operative prognosis in localized peritonitis depends upon the seat of infection, the promptness with which surgical measures are resorted to, the technic observed in the operation, and our ability to reach the cause.

It is impossible to give the operative prognosis of a diffuse septic peritonitis until surgeons adopt some uniform classification; we cannot presume to deny that it is curable, yet such authors as Senn, Treves, Tietze, question that these cases are subjects for surgical intervention.

The prognosis in suppurative peritonitis becoming general is much influenced by the measures employed for the removal of the septic material and provision for drainage.

CONTRAINDICATIONS TO OPERATION.—An established acute peritonitis is a surgical affection, yet this axiom does not imply that the propounders of it authorize immediate intervention regardless of condition. In acute localized peritonitis which is evidently waning, operation should be watchfully deferred; functional recovery may render operation unnecessary; or, as in chronic appendicitis, with the subsidence of inflammation, operation may be undertaken under more favorable local and general conditions. Should a circumscribed abscess form, the adhesions are more protective than any means at the surgeon's command, and their mere presence signifies bacterial impotency, a comparatively safe field in which to work. Patients suffering from perforation may evidence such a degree of shock as to restrain any prudent operator; if there is no reactionary response, operation will only hasten the end. Complicating conditions may in themselves be sufficiently grave to cause death, and their presence may contraindicate any attempt to relieve the peritonitis.

TREATMENT.—The fact that peritonitis almost invariably occurs as a complication of some other process and not independently, has been definitely brought out in these pages. This fact gives the keynote to what should be the treatment of peritonitis, prevention. The primary focus, whenever and wherever possible, should be so handled as to anticipate the spread of infection to the peritoneum, for our belief is that no treatment, medical or surgical, of an established infection has been proved of definite value, particularly in the severer types of infection, such as that due to the streptococcus,

the common colon bacillus, etc. Where infection has taken place, our efforts must be limited in most instances to cutting off the source of infection, and we must to a great extent leave to nature's efforts the handling of the process already actually inaugurated. In such cases the removal or sequestration of the primary focus wherever possible with the greatest promptness must be our first object in treatment. No plan of treatment that does not make this the first consideration is worthy of a moment's thought. If we will keep this fact definitely in view, we can readily make our choice of the measures proposed for the treatment of this condition.

All plans for the medical treatment of peritonitis fall into one of three classes, more or less accurately represented by the Alonzo Clark, Tait, and Ochsner methods.

First we have the idea of specific medication, a type of which is the Clark method, which was based upon the belief that opium was a specific antidote for peritoneal inflammation, and consisted in the administration of enormous doses of this drug. One case reported by Flint as being under the care of Clark himself took over a thousand grains of opium in one week and recovered. No one at the present day has any belief in the value of opium as a specific. The good done by this drug is in checking peristalsis, thereby preventing the spread of infection, acting as a mild stimulant, relieving pain and nervous symptoms, and making respiration easier and more satisfactory. The fact that it inhibits leucocytosis and so lessens phagocytosis may possibly offset all the good it does, but it does seem to be almost essential at times, in post-operative handling of cases, for the relief of symptoms. In small doses hypodermically it does much good in this way, and the relief accorded not infrequently changes the whole aspect of the case for the better.

Of the other two plans of medical treatment each follows out one of nature's methods for the cure of peritonitis, Tait by aiding the elimination of the poison and Ochsner by attempting to localize it.

Tait by the administration of repeated small doses of saline purges hoped to eliminate the infection through the bowel, you might say, by establishing drainage in that way. As a post-operative plan of treatment, and it was so used by its originator, it is intelligent and satisfactory, though with the exception of one class of cases it would be homicidal to use it without first removing or sequestrating the focus from which the infection started, pro-

vided that focus lay within the abdominal cavity. The one exception referred to is gonorrheal peritonitis. Acute gonorrheal peritonitis whether localized or generalized is a medical disease, and should be treated by saline purgation, hot applications, and when pain and nervousness demand it, morphia hypodermically.

Ochsner by starvation, gastric lavage and low enemata keeps the stomach quiet, sets peristalsis at rest and empties the colon. To this extent he aids in localization. In cases of peritonitis, especially of appendiceal origin (it is peritonitis complicating appendicitis for which Ochsner recommends his method), *when the patient refuses operation,* and in most cases of hæmotogenous peritonitis, it is probably the best plan of treatment. It cannot, however, when the primary focus is within the peritoneal cavity, take the place of intelligent surgical handling of that focus nor is it altogether as rational and satisfactory a plan of post-operative treatment as Tait's. When this plan is used the nutrient enemata recommended by Ochsner can be left off without detriment to the strength, and with great increase in the comfort of the patient.

As already emphasized, there can be no doubt that the most satisfactory surgical treatment of peritonitis is preventive. When for any reason this preventive treatment has not been instituted in time, we are confronted with the question of dealing with two conditions, the primary disease and the complicating peritoneal sepsis. As to the first, when the focus is in the peritoneal cavity, there is no difference of opinion. When there has been no localization by the formation of limiting adhesions, the primary focus must be removed or sequestrated as the first step of operative treatment in order to protect the peritoneum from further soiling. We cannot place too much emphasis upon the fact that we must not persist in efforts to do complete surgery. However desirable it might be to remove entirely the disease at the primary focus, we cannot run the risk of prolonged operation with manipulation of peritoneal surfaces in doing this. We must keep in mind that the principal object of surgical interference in peritonitis is to prevent the addition of further infection to the peritoneum. This must be done in the shortest time, and with the least possible amount of manipulation, and all other considerations are secondary to this. On this basis there is little indication for peritoneal surgery in peritonitis of hæmotogenous origin unless there has been localization with

abscess formation. With localization of the process, and the formation of an abscess cavity, often practically extra-peritoneal, the propriety of removing intra-abdominal primary disease depends on whether or not this can be done without too much risk of soiling the general peritoneum as a result of the breaking up of limiting adhesions. Here our first consideration is to drain the abscess and get rid of the acute infective process. If necessary or advisable, the primary disease can be removed later and under much more favorable conditions.

With generalized infection after removal of the primary disease, the question of how to deal with the peritoneum itself comes up. Shall we or shall we not make a general toilet of the peritoneum? As has been shown clinically by Murphy and others, and explained on bacteriological grounds by Dudgeon and Sargent, in most cases of generalized inflammation the peritoneum can take care of itself if not mishandled at the time of operation. When the white staphylococcus is present and doing effective work through the exudate of leucocytes and plastic material that it excites, a general toilet of the peritoneum either by flushing or wiping is hardly short of malpractise. When the colon bacillus is present alone, or when the streptococcus or bacillus pyocyaneus is present, the outcome is almost necessarily fatal with any or no treatment. It is possible that in these last types of cases flushing might be beneficial. The exudate contains neither plastic material nor active phagocytes, and no harm can be done by washing it away. If this is to be done a free incision should be made in the median line in order to allow easy access to all parts of the cavity. Only in this way can lavage or flushing be satisfactorily carried out. The extreme difficulty, not to say impossibility, of determining the character of infection at the time of operation makes the selection of cases for lavage a hazardous matter, and in the present state of our knowledge the use of irrigation in the treatment of peritonitis had best be abandoned. In this connection it might be well to state that there is no contraindication to the use of irrigation for the purpose of removing foreign material after perforating gastric ulcer, etc. Our object here is the prevention and not the treatment of peritonitis.

Enterotomy: Enterotomy, enterostomy and intestinal resection can have little place in the treatment of peritonitis itself, though

they may be indicated for some condition of the bowel complicated by peritonitis. Thorough lavage cannot be done without evisceration when the bowel is distended, and after evisceration under such circumstances, enterotomy will often be needed to enable the operator to get the bowel back into the cavity. If there should prove to be a class of stages in which lavage is indicated, enterotomy will be a necessary adjunct to this treatment.

Drainage: After operation done for abdominal disease complicated by peritoneal infection, shall we drain? Only when it is impossible to remove all necrotic, or intensely septic tissue. Here our object is the sequestration of a small area rather than drainage of the cavity, and for this purpose we use gauze either plain or in the form of a cigarette drain. Sequestration of an area is all we get in any instance by abdominal drainage, and only when sequestration is demanded does it pay for the weak spot left in the belly wall. A thorough local toilet by sponging is better except under the circumstances mentioned above. In sponging the peritoneum every possible care must be exercised to avoid removing the epithelium, as the ultimate disappearance of peritoneal exudates depends on its preservation. Adhesions are certain to result from plastic exudate when the epithelium is removed. In local abscess formation, with destruction of the peritoneal epithelium, there is no chance for absorption of the infection, and drainage is imperative. Here tube drainage should be used, as it really drains and is much less painful to the patient in the after-handling of the case.

Fowler's Position: The general use of Fowler's position in the after-treatment of peritonitis demands mention of it here. Many patients are more comfortable in this position, many are not. Its principal justification is the belief that exudates gravitate into the pelvis from the general cavity, and are evacuated by the tube drain usually placed there. However that the general cavity is drained in this way is unproved.

The introduction of a solution of nucleic acid, horse serum or salt solution before closure of the operating wound to produce leucocytosis is of doubtful value. The use of normal salt solution by hypodermoclysis, intravenous infusion or in the way of a drip enema is frequently indicated for stimulation. The last-named method is the simplest and the least painful and is very satisfactory for ordi-

nary use. In the severest cases intravenous infusion will be demanded.

The use of sera in the after-treatment of peritonitis is a matter for future development and must be based on the production of sera especially adapted to the bacteriology of the given case.

Chronic Peritonitis (Non-Tubercular): It is manifestly improper to describe as examples of chronic peritoneal inflammation cases where adhesions and encysted exudate exist after acute infections. This has been frequently done by writers with no effort made to prove the persistence of an inflammatory process. There are true chronic inflammatory processes in the peritoneum, however, and several pathological types have been accurately described. These conditions have been designated:

1. Chronic Exudative Peritonitis.
2. Chronic Adhesive Peritonitis.
3. Chronic Hyperplastic, or Proliferative Peritonitis.
4. Chronic Hemorrhagic Peritonitis.
5. Chronic Carcinomatous Peritonitis.

Chronic Exudative and Chronic Adhesive Peritonitis: These two types differ only in the amount of plastic material found with a serous or seropurulent exudate always present. The etiology and particularly the bacteriology of these conditions have not been worked out, but the process is supposed to be a mild infection starting from some visceral focus. Sufficient tests with negative results have been made with tuberculin and by the injection of animals with the exudate to exclude tubercular infection.

SYMPTOMS.—The onset is gradual. The development of ascites or colic due to adhesions may first attract attention. There is progressive involvment of the patient's health with the loss of strength and the loss of flesh. Spontaneous pain is not always present, but more or less tenderness can be elicited by pressure. Fever is present in about one half of these cases.

Chronic Hyperplastic or Proliferative Peritonitis: In this type the involved peritoneum loses entirely its normal appearance. Connective tissue hyperplasia takes place and there is substituted for the peritoneum over a more or less extended area a dense gristly connective tisssue which tends to contract and may lead to a diminution in the size of the viscera with shortening of the mesentery and contraction of the omentum into a fibrous cord. Serous effu-

sion is common, being due to a great extent to portal obstruction resulting from involvment of the gastro-hepatic omentum. The process is local at first, frequently involving the liver primarily, but tends to spread progressively. As with the exudative and adhesive types, other serous membranes, the pleuræ and pericardium, may be involved.

SYMPTOMS.—The development of ascites with digestive and obstructive symptoms and progressive deterioration of the patient's general health are the manifestations of this condition. The thickened omentum may be palpated after paracentesis.

Chronic Hemorrhagic Peritonitis: According to Osler, this condition was first described by Virchow. It is usually confined to the pelvic peritoneum, and is marked by the formation of thin layers of richly vascularized connective tissue from which hemorrhages occur. This type is a pathological curiosity.

Carcinomatous Peritonitis: This condition is occasionally primary, but is usually secondary, particularly to cancer of the stomach or ovaries. It is sometimes marked by a diffuse fibrous thickening of the peritoneum similar to the hyperplastic condition already described, and from which it can only be differentiated by microscopical examination.

SYMPTOMS.—In the absence of evidence of primary cancer of the stomach or some other viscus, peritoneal cancer may be marked merely by cachexia and ascites. Digestive and obstructive symptoms will sometimes be present, and tapping may result in the discovery of nodules in the peritoneum.

Diagnosis of Chronic Peritonitis: A clinical differentiation between the different forms of peritonitis is difficult and a differentiation of these types from tuberculous peritonitis equally so without the tuberculin test or the injection of the exudate into animals. Advanced age of the patient, rapid progress of the disease, pronounced cachexia and a hemorrhagic or chylous exudate points to cancer. Cirrhosis of the liver with which chronic peritonitis is more than apt to be confused can only be excluded when there is an absence of alcoholic or specific history and of marked digestive symptoms with hematemesis and melena.

Treatment of Chronic Peritonitis: In the chronic exudative and adhesive types incision and irrigation are indicated. Lennander recommends repeated irrigations if the first is not successful. Good

may be done by breaking up adhesions or at least by cutting out bands of adhesive material. Resection of badly involved portions of the alimentary tract that lend themselves to the procedure is indicated. In chronic proliferative peritonitis and in carcinomatous peritonitis nothing can be done beyond tapping when the ascites become excessive.

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TUBERCULOUS PERITONITIS.

DEFINITION.—A specific inflammation of the peritoneum excited by the bacillus tuberculosis.

FREQUENCY.—König from post-mortem examinations concludes that tubercular deposits are found in about 5 per cent. of all autopsies. In tuberculous subjects there is peritoneal involvement in 20 per cent. of cases (Wothen); according to Borschke it is something less than 16 per cent. Of the 107 cases of tuberculous peritonitis in König's table, 99 had coexisting phthisis. In practically all cases a primary focus is found.

ETIOLOGY.—**Age:** It is most frequently met with clinically between the ages of twenty and forty years, yet there are no limitations of age. In 1892 Hartmann and Aldibert collected 18 cases bacteriologically proved in children. Kissell says that it is far more common in children than is supposed. Holt asserts that the peritoneum is quite frequently the seat of tuberculous inflammation in early life; 7 of his 56 cases were under 3 years of age. In 883 collected autopsies upon tuberculous children Biedert found the peritoneum involved in 18.3 per cent., paralleling in frequency its occurrence in adults.

Sex: Statistics taken from operations show that tuberculous peritonitis occurs twice as frequently in women as in men. Autopsical findings would indicate that it is more common in males. Cheyne has alluded to this discord. As both sexes are equally exposed to infection, there is no physical reason why it should show a predilection for one sex over the other. It is found at operation in women more frequently, since more of that sex are laparotomized.

Malnutrition, exanthemata, pertussis, and intestinal catarrh are said to affect the incidence of peritoneal tuberculosis. Kelly regards parturition as predisposing to it. Hepatic cirrhosis, alcoholism, and tuberculous peritonitis are concomitant phenomena without an established sequence.

The neoplastic formation upon the peritoneum, the tubercle, is here, as elsewhere, immediately and solely due to a localization

of the bacillus tuberculosis. The bacillus tuberculosis reaches the peritoneum from a primary focus, usually, by the blood or lymphatics; occasionally, this serous membrane is directly infected by the escape into it of tubercular matter. Acute miliary tuberculosis with its wide dissemination along the courses of blood-vessels indicates a hematogenous origin; such patients are the subjects of general tuberculous infection. Peritoneal tuberculosis is commonly secondary to primary thoracic lesions in the lungs, pleuræ, or lymphatic glands; Merkel calls attention to the frequency with which the primary focus is in the glands at the bifurcation of the trachea. The route of infection is probably by lymphatics. Infection of the peritoneum from the thorax through the diaphragm may occur by continuity or by ulceration. Tuberculosis of intestinal origin occasionally occurs from ulcerative lesions in the intestinal mucous membrane; or as Allchin asserts, infection may take place without any recognizable *atrium*. In such cases the virus reaches the mesenteric glands, subsequently involving the peritoneum. Osler considers tuberculous peritonitis in women a common sequence of tuberculous salpingitis. Upon the other hand, Williams asserts that tuberculosis of the Fallopian tubes is far more frequently the result than the cause of peritoneal tuberculosis. That infection from without through the genitals is a common mode of invasion seems established. In men genital tuberculosis is rarely primary. The etiology of the associate genital and peritoneal tuberculosis in the male is often a perplexing problem to solve.

PATHOLOGY.—The deposition of a peritoneal tubercle excites a cellular activity reactionary or defensive in character, which constitutes in part the phenomenon of tuberculous peritonitis. Each tubercle is surrounded by a zone of new-formed cells, which show a tendency to form a false membrane. This zone is usually marked by an area of hyperemia with strong disposition to hemorrhage, in which setting the minute nodule is found. Often there is macroscopically no surrounding inflammation.

In local infections the area involved may be very limited, or discrete tubercular nodules may appear over a greater or lesser surface. The most common sites of local infection are the pelvic peritoneum, appendiceal region, under surface of the diaphragm, and hernial sacs. In general miliary tuberculosis there is wide

dissemination of the tubercular deposits over the visceral and parietal peritoneum.

In conjunction with the deposit of tubercular matter in the peritoneum there occurs a serous exudation; in acute cases it is often hemorrhagic. Peritonitis, whether common or of this specific form, has as an essential part of its pathological process lymphangitis, with consequent lymph-thrombosis. The serous exudate is great or small as expressive of the acuteness and extensiveness of the lymphatic obstruction; hence in those cases of acute miliary tuberculosis general to the peritoneum, copious and unconfined serous effusion occurs. In the subacute forms the absorptive function of the peritoneum is only gradually lost; and while the exudate may be ultimately as great, it is more gradual in accumulation and often associated with plastic adhesions, confining the fluid effusion to the area involved.

In chronic peritonitis the changes that occur must be considered as anatomical evolutions in the life-history of the tubercular granulation. The separate tubercles are solid and are surrounded by a pigmented area. As tuberculous peritonitis is a secondary infection, the attenuated germ begets a fibrinous inflammation; and this is the striking feature of peritoneal tuberculosis, the tendency to fibrosis with sequestration and absorption of the tubercle; but should degeneration occur, that is, caseation of the granulation-tissue, masses of soft, cheesy consistency are formed, and with multiple infection there is puriform degeneration.

In the acute forms of tuberculous infection the peritoneum loses its pearly luster; it is red, granular, thickened, and hemorrhagic. With general involvement there is rapid accumulation of fluid and great distention.

In subacute cases with mild and progressive infection there is displacement and anchorage of viscera, welding of opposing peritoneal surfaces, and effacement of visceral outlines; the walls of the viscera are thickened and friable. The normal peritoneal pockets may be effaced, or else distended with effusion. Between the coils of intestines, viscera and parietes, in the recesses formed by false adhesions, loculi containing exudates are found. The mesentery is thickened, and, contracting, displaces the intestines. The omentum, infiltrated with tubercles, contracts and

rolls into a mass which lies athwart the abdomen; or else from acquired adhesions acting as a partition, it may exclude the infected area. The fibrous formation, a stage in process of peritoneal tuberculosis, may be accompanied by effusion; or in other cases the peritoneum may be quite dry. Caseation is always attended by fibrinous deposit. In children especially, the parietal peritoneum becomes very thick and resisting—leather-like. Mesenteric glands may be enlarged but not necessarily caseous. Thoracic glands and pleuræ most constantly show the tubercular changes. Tuberculous pericarditis is unusual.

The morbid changes are determined by the mode of infection, the virulence of the poison, the individual's resistance, and frequently by the occurrence of a mixed infection of intestinal origin. If we bear in mind that the morbid changes are incident to an infection which may reach the peritoneum in any part of its wide area and in varying potency at different times, it will necessarily appear that the picture presented will not be uniform. An abdomen may reveal an area of recent invasion with discrete or confluent tubercular granulations, concealed by an encapsulated serous or sero-sanguineous effusion; in another part the tubercles are engulfed in the fibrous changes consequent upon the adhesive inflammation; and still elsewhere, at the seat of invasion and oldest infection, caseation with ulceration may be going on. A pathological classification offered by Aldibert and generally accepted by authors, based upon the conspicuous or prevailing feature, comprises the *ascitic, fibrous,* and *ulcerous* varieties. Aldibert makes many unnecessary subdivisions of these heads. Caillé, and after him many others, confuse this simple pathological classification with a clinical variety styled *tubercular peritoneal tumors.* Every phase of tuberculous peritonitis may very properly fall under the three heads.

1. **Ascitic:** In acute miliary tuberculosis there is a wide-spread deposition of tubercles over the peritoneum, accompanied by an abundant serous or sero-sanguineous exudate. These patients are succumbing to general tuberculosis, and are not subjects for operative consideration. In the less acute or chronic invasion there is a copious fluid exudate rich in albumin, transparent, brown or red from admixture of blood, or turbid with fibrin, exceptionally purulent, and of high specific gravity. It may be

unconfined or limited to areas by adhesions. In the most chronic cases encapsulated effusions, one or many, are generally found. The prevailing feature in these ascitic cases is the abundant effusion, the absence of strongly organized adhesions, and the conspicuous appearance of the tubercular nodules.

2. **Fibrous**: The inflammation excited by the bacillus tuberculosis may be adhesive from the first, or the more acute forms may become fibrous. In advanced cases the tubercular nodules have disappeared or are concealed by the new connective-tissue formation. The intestines are displaced and anchored; broad bands of adhesions unite the adjacent coils; other parts of the intestinal peritoneum, more acutely affected, may be red and covered with more or less closely adherent flakes of yellow fibrin. All visceral outlines are obscured; and the abdominal organs have the appearance of plaster casts. The parietal and visceral layers of the peritoneum are thickened and leather-like; the normal peritoneal pockets are effaced; the omentum, abnormally attached, is stretched into a coil or roll, or else thickened, friable, and contracted upon itself; it lies as a hard tumor across the upper part of the abdomen; the mesentery, perhaps enclosing cheesy glands, is contracted and shortened, frequently drawing the intestines to the right; there may be no fluid whatever, but these dry adhesions practically obliterate the peritoneal cavity; or else encapsulated effusions, either serous or purulent, with massive fibrinous adhesions and agglutinated omentum and intestines, may form one or multiple tumors distinctly palpable and confusingly misleading in diagnosis. The nodules formed by connective tissue and degenerating tubercles are very large, and sometimes appear as pendulous tumors two centimeters in diameter (MacCallum). In these extreme cases of fibrosis all effusion has disappeared, and it seems as though nature is attempting obliteration of the peritoneal sac. In some instances the fibrous tissue appears to spring exclusively from the mesentery; in a recent case the author found cheesy degeneration of mesenteric glands making a mass larger than one's fist, and from this as a center, broad bands of fibrous tissue enclosing cheesy matter radiated in different directions, around, under, and over which the intestines were almost hopelessly entangled.

3. **Ulcerous**: A caseating tubercle, from its tendency to destroy by ulceration the walls of the intestines, producing suppuration and fistulæ, gives rise to the ulcerous form of tuberculous peritonitis. By caseous changes in the urachus, or by adhesions of a suppurating area to the anterior parietes, edema and inflammation of the abdominal wall is noticed, and occasionally a purulent or fecal discharge from the umbilicus appears.

Caseation may occur in an abdomen in which there are areas of fresh tubercles. In others there is a curious medley of ascitic, fibrous, and suppurative peritonitis. This, as all other forms, may be local or general to the peritoneum. The fibrous tissue, of every degree of density and thickness, is a constant morbid change accompanying the caseation. While it is no longer denied that the tubercle-bacilli are possessed of pyogenic properties, yet when there are puriform accumulations mixed infection is frequently found.

Ulceration of the intestinal mucous membrane accompanies the acute forms of miliary tuberculosis; it is not often found in the more chronic types of tuberculous peritonitis. Glandular enlargement and caseation of the thoracic, mesenteric, and retro-peritoneal glands, especially the retro-peritoneal pelvic glands, pleurisy, and pulmonary phthisis, latent or active, are common post-mortem findings.

Symptoms.—**History**: A patient, the subject of tuberculous peritonitis, is frequently a descendant of tuberculous parentage. We are often able to establish some causal relation between the peritoneal eruption and some recent illness; pleurisy, pneumonia, inflammation of the internal genitalia, and in children the exanthemata, appear to render them susceptible to the invasion of the bacillus tuberculosis. Obscure abdominal pains, digestive disorders, anorexia, diarrhea or constipation, irregular fever, and progressive loss of weight, are symptoms of established importance. But we are so frequently confronted with peritoneal tuberculosis in individuals of robust health that too much consequence must not be attached to the history and previous condition. Abdominal operations have shown that peritoneal tuberculosis may be very latent. On the other hand, the onset may appear suddenly and violently, and be marked with such acute symptoms as to be mistaken for ileus or appendicitis. In other

cases, especially in acute miliary tuberculosis, the febrile symptoms, gradually intensifying, may simulate typhoid fever; Osler repeatedly refers to this common error.

Associate Lesions: If it is correctly held that tuberculous peritonitis is a secondary affection, a great aid to its diagnosis is the detection of the primary focus. No examination is complete that fails to take in review the entire patient, directing especial care to the investigation of the lungs, pleuræ, genitals, rectum, and colon. A preëxisting, concomitant, or secondary pleurisy contributes greatly to the value of other signs.

Temperature and **Pulse:** There is no characteristic temperature-chart in tuberculous peritonitis, nor can there be positive assertions made that the temperature is indicative of the stage and behavior of the disease. An acute disseminated peritoneal tuberculosis, suddenly appearing in one of robust health, may terminate within three weeks, and persistently low or subnormal temperature mark the whole course of the disease (Halstead). A suppurative condition with large collections of encapsulated purulent matter is not necessarily attended by fluctuating temperature. Osler calls especial attention to subnormal temperature in the chronic forms of tuberculous peritonitis. Persistent temperature with evening exacerbations is usually noted in the acute and subacute forms of the ascitic variety. As the condition becomes more chronic the temperature declines. With caseation, especially when there are purulent accumulations, fluctuating temperature and sweats are usual. When the deposit is restricted to the pelvis by adhesions there is often an absence of pyrexia.

The pulse is influenced by the general toxicity, the degree of marasmus, the amount of distention, and the acuteness of the course of the disease.

Digestive Disturbances: The onset, especially in children, may be attended by vomiting, and this symptom may become so conspicuous in individuals of any age that, when coupled with the phenomena of distention and abdominal pain, we may wholly misinterpret its cause. In gradual infections there is loss of appetite. The bowels may be constipated, or diarrhea may be present. A mucous diarrhea is frequently due to intestinal ulceration, and suggests an active process in parts other than the peri-

toneum. In chronic fibrous peritonitis constipation is the prevailing condition. In these cases prolonged constipation, relieved by a spontaneous diarrhea, the progressive wasting, and the presence of a tumor suggest intestinal carcinoma.

Pain: In very acute cases the onset may be marked by violent pain. In fibrous peritonitis, pain, colicky in character and often severe, is due to the fixation and constriction of the intestines, requiring exaggerated peristalsis. In the ascitic and complex forms in which there is great distention, local or general, actual pain is not so pronounced, but there is great discomfort and abdominal heaviness, loin and sacral pains. Kelly emphasizes the value of pain on urination in women as a symptom of peritoneal tuberculosis. Pain is often caused by intestinal distention and relieved by free purgation. Respiration is often restrained because of pain on movement of the diaphragm and by reason of the associate pleurisy.

Menstrual Disturbances: In the early stage of tubal tuberculosis dysmenorrhea and metrorrhagia are common symptoms. With the progress of the disease and decline in the general health amenorrhea supervenes.

PHYSICAL SIGNS.—**Inspection:** The general appearance of the patient may in itself be suggestive. The entire integumentary surface may be pigmented, probably due to pressure upon the adrenals by enlarged mesenteric glands; this symptom of pigmentation has been noticed without tuberculous involvement of the adrenals. In alcoholics with hepatic cirrhosis jaundice may be noticeable. In very acute cases ecchymoses and petechial spots have been observed. In the forms accompanied by effusion the abdomen is distended, usually symmetrically so in the more acute cases; irregularly enlarged when there is sacculation or localized infection. If there is great distention accompanied by emaciation the surface veins of the abdomen may be very conspicuous. The umbilicus is pouting; when suppuration is present there is an area of redness and edema surrounding it, and occasionally a discharge from it, serous, purulent, or fecal—this sign is regarded as pathognomonic. In cecal and appendiceal tuberculosis the distention is confined to that region. In chronic tuberculous conditions the abdominal distention is often due to tympany and is variable. In fibrous peritonitis the abdo-

men may be retracted, especially in its lower zone; and when partial intestinal occlusion occurs the exaggerated peristalsis is evidenced in visible patterns. The lower extremities in the latter stages become edematous; the swelling may be due directly to pressure from massive mesenteric tuberculosis.

Palpation: An acute infection with disseminated peritoneal tubercles is with rare exceptions attended by great effusion. The belly is smooth, elastic, fluctuant; the resistance is generally greater on the left side. In the more chronic cases the effusion has either disappeared or has never been a conspicuous feature; or if present it is encapsulated, yielding under palpation areas of unequal resistance with circumscribed elasticity or fluctuation. The boundaries of these circumscribed collections of fluid are irregular, ill-defined, and often incomplete; the fibrous adhesions uniting the intestines, omentum, parietes, and viscera form the limiting walls. The conspicuousness of the swelling and its palpability are conditioned largely upon the degree of tympany. In the most chronic forms, *caseous* and *fibrous,* irregular doughy tumors may be felt in any portion of the abdomen. The omentum, contracted, thickened, and infiltrated, forms one of the most characteristic signs in tuberculous peritonitis. The massing of intestines in dense bundles of fibrous tissue, the whole enclosing serous, caseous, or purulent matter, may form single or multiple tumors in any portion of the abdomen. In a recent case of dry fibrous peritonitis a tumor as large as an orange, visible and palpable, was due to caseous mesenteric glands. In children especially, the abdomen is tender in the acute forms. In all individuals tenderness, inconstant in location, may occur with recrudescence or from intestinal complications. In the fibrous stage there is often an insensitive condition of the abdomen. Pain may not be elicited in patients with tuberculous peritonitis by firm and deep pressure, but upon withdrawal of the hand and sudden expansion of the abdomen children will cry out with pain. In thin subjects, especially in children, the thickened and stiff parietal peritoneum may be recognized by rolling it between the palpating fingers. Crepitus may be both felt and heard in the fibrous variety. Gurgling sensations, due to intestinal stenosis or to shifting effusion, are more common. While fluctuation is common in the acute forms, it is

not elicited when the disease pursues a more chronic course, either because of the absence of effusion or because of its loculated state. With suppuration there is edema, a doughy resistance, and increased tenderness of the parietes, most usually found in the umbilical region.

Vaginal examination, especially when the infection proceeds from this quarter, reveals a fixed uterus, a resisting indurated vault, the appendages cannot be easily outlined, and nodular, sensitive masses are felt. In males rectal examination may show pelvic glandular infiltration with great sensitiveness. Any tumor below the umbilicus can, by combined rectal and abdominal palpation, be explored, and its density and thickness estimated.

In hernial tuberculosis the empty sac is thickened, resisting, and irregular; it refills quickly after emptying and there is no gurgling on taxis (Andrews). The spleen is palpably enlarged.

Percussion: In the ascitic variety the difference in the percussion-note, obtained respectively upon the left and right sides, is significant; a gradually increasing dulness as we percuss from right to left is not pathognomonic but quite significant; the rational explanation is that the contracting mesentery draws the intestines to the right. Areas of percussion-dulness may be found at the seat of any limited infection. It is in the *chronic caseous variety* with tumor-formation that we find percussion most valuable. These tumors are cakes; when composed of matted lymph, omentum, and intestines, sacculating ascites or pus, they are usually superficial and rest in apposition to the parietes. If operation is contemplated it is important to distinguish them from cheesy glands, which underlie the intestines and produce palpable tumors, tympanitic upon percussion. It must be borne in mind that the physical characteristics of intra-peritoneal tubercular tumors are susceptible of sudden changes.

Exploratory Incision: All symptoms and signs may be inconclusive, and diagnosis will be made only by an operative exposure. The macroscopical picture is generally definite, and is confirmed by microscopical demonstration. In the ascitic form and in the advanced suppurative cases tubercle-bacilli may not be found in the effusion. Fibrous tuberculosis may be mistaken for carcinoma and papilloma of the peritoneum.

CLINICAL TYPES.—The clinical course which a tuberculous peritonitis pursues is either *acute, subacute,* or *chronic,* a division based not only upon the duration but also upon the symptomatic behavior.

Acute Tuberculous Peritonitis: In acute miliary tuberculosis the peritoneal eruption may be only incidental to a more active focus, or it may constitute the predominating feature. And while effusion characterizes this pathological course, the rapid wasting, the hectic condition, the pyrexia, failing pulse, diarrhea, and abdominal symptoms may all suggest merely an enteric fever. These cases are not surgical in nature and concern us only in the differential diagnosis.

Subacute Tuberculous Peritonitis: A more gradual onset, with steady wasting, slight pain, progressive distention, localized or general, subnormal temperature with evening pyrexia, moderately frequent pulse, are symptoms usually associated with general or at least extensive tuberculous infection, producing a serous effusion with moderate amount of fibrous formation. If these cases pursue a more protracted course, pain becomes more conspicuous, constipation pronounced, localized areas of dulness appear, tumors are formed, tenderness is marked, emaciation progressive, while the temperature may still be an inconspicuous feature. The suspected pathological state in such cases is a sero-fibrinous peritonitis, with depositions of lymph and intestinal adhesions.

Chronic Tuberculous Peritonitis: Chronic peritonitis may be of the fibrous variety, and is exhibited in patients thin and gaunt, suffering with digestive disorders, but not necessarily with disturbance of temperature or pulse. The state of marasmus is due chiefly to visceral displacement and functional inhibition. Pain, however, may be a persistent and distressing feature. There is slight or no effusion, yet areas of dulness, due to fibrous thickening and massing of viscera, may be found. Sudden tympany, and paroxysmal pain and vomiting, attributable to partial intestinal obstruction, signalize and complicate the clinical course. Great emaciation, fluctuating temperature, often high, frequent and weak pulse, profuse sweating, variable tympany, local tumors, hard or elastic, pain and tenderness, peri-umbilical edema and redness, possibly a purulent or fecal fistula, swelling of the

extremities, are symptoms associated with caseous degeneration and ulceration.

DIFFERENTIAL DIAGNOSIS.—**Appendicitis:** Exceptionally the onset of tuberculous peritonitis is so peracute, without even the suggestion of a prodrome, that the stormy attack is mistaken for appendicitis. Musser refers to such cases, one operated upon by Keen; but in this particular case the diagnosis was not in fault, a perforating tubercular ulcer of the appendix being found. The patient recovered from the operation to die in two months from general tuberculosis. Brunn has twice mistaken tuberculous peritonitis for appendicitis. Localized tuberculous cecitis and appendicitis, with caseous changes, have on two occasions led the author astray.

Enteric Fever: In the symptomatology we have referred somewhat in detail to the differential confusion existing between general miliary tuberculosis and enteric fever. This error arises chiefly through imperfect examination. In miliary tuberculosis the active pathological foyer, in connection with or remote from the abdomen, should be discovered. The sputa and urine will show the bacilli tuberculosis; and in enteric fever Widal's reaction may be obtained.

Intestinal Obstruction: Lejars says that the severity of the onset may simulate intestinal obstruction. These cases must be very rare, indeed. Fibrous tuberculosis may pursue a very masked course, when suddenly the symptoms of obstruction arise. The error is not one of present condition but of causative morbidity, and is entirely pardonable.

Ovarian Cyst: Sacculated adhesions resembling an ovarian cystoma cause the most common diagnostic error. To Spencer Wells's error we must acknowledge our indebtedness for the discovery of the surgical treatment of peritoneal tuberculosis. The general disturbance that attends sacculated tuberculous peritonitis, the location of the tumor, which is usually in the middle zone of the abdomen, though it may be in the pelvis, occasionally in the upper part; the history of the protracted illness and wasting, often antecedent to any abdominal phenomena; and the physical signs of palpation and percussion, are usually sufficient to prove discriminative, yet few surgeons of wide experience are guiltless of this error.

Chronic Peritonitis: Here the differentiation can only be made in some instances by the tuberculin test, or by aspirating and injecting the exudate into animals. Even at operation the macroscopic evidences may not be sufficient for positive differentiation; especially is this true of the fibrous form of tuberculous peritonitis. Unmistakable evidences of tubercular foci outside of the abdominal cavity, of course, are very suggestive, not to say positive indications, as to the character of an obscure chronic peritonitis.

PROGNOSIS.—Prior to König's first report, in 1884, tuberculous peritonitis was regarded as an almost uniformly fatal disease when treated medically. The effects of the operative treatment have been to teach us that peritoneal tuberculosis is often a latent and localized affection which may even run its course without producing special symptoms, and with a natural tendency to recover more often than is even now conceded. While the prognosis is not altogether bad, the gravity of an acute peritoneal infection, with or without recognizable primary lesion, cannot be overestimated. If we hold that peritonitis of this special variety is secondary to visceral or glandular infection, the peritoneal involvement may be arrested or controlled, but the ultimate prognosis will rest upon the activity of the primary lesion.

Peritoneal tuberculosis, appearing as a part of a general miliary tuberculosis, offers but little hope for even a temporary arrest. An acute peritoneal involvement, though localized, may become rapidly general and be attended with such toxic symptoms that we should regard it with the greatest apprehension.

While König taught us the value of the operative treatment, he was among the first to advocate a selection of cases, and to condemn laparotomy as a uniform procedure applicable to all types of peritoneal tuberculosis. Medically treated this affection is amenable to cure. Schroeder in his medical clinic obtained about 41 per cent. of cures. Rose, who cites articles by Borchgrevink who strongly advocates expectant treatment over surgical measures, gives these rather startling statistics: of 44 cases of tuberculous peritonitis 22 were treated by operation, with 64 per cent. of recoveries; 22 without operation, with 82 per cent. of recoveries, 68 per cent. of which were of over a year's standing. Späth long ago advocated conservatism, but

not to the exclusion of surgical treatment. Caillé, in a strong article, shows the futility of medical treatment of tuberculous peritonitis in children.

The operative results give a percentage of recoveries varying from 25 to 80. That absolute anatomical cure may follow the surgical treatment is proved by numerous instances subjected to a second abdominal section for various causes, showing the peritoneum clear of all effects of the previous tuberculous inflammation. Cheyne says that in 385 operations 53 per cent. of the patients were well two years after the operation. Rörsch presents the statistics of 358 cases operatively treated, 250 of which were cured, 70 per cent.; 118 were under observation six months, 79 one year, and 53 two years. The immediate operative mortality is very slight, not over 3 per cent.

Tuberculous infection of the parietal wound does not occur. Pyogenic infection is infrequent. A complication to be greatly feared is the formation of a fecal fistula; it will not occur in the ascitic form unless drainage is employed; it is especially liable to follow in the ulcerous form. The fistulæ are very obstinate; in Czerny's clinic in 3 out of 4 cases he failed to close the fecal fistula by suture; the author has had the same mortifying experience in 2 cases.

The subacute and chronic ascitic forms are the most amenable to operative treatment. In only exceptional instances of complications and for the relief of special conditions should operation be undertaken in the fibrous form. The ulcerous form offers an unfavorable outlook; yet even these cases may eventuate in cure. After five different operations upon one patient, extending through a period of two years, the author had the satisfaction of seeing absolute cure follow from drainage of many caseous masses; the patient is now a woman grown, in perfect health and the mother of children.

All forms except the peracute are at least temporarily benefited, and a very conservative estimate of absolute cures can be placed at about 50 per cent. It will be a very difficult matter to eradicate from the active surgeon's mind these established benefits of the operative treatment of tuberculous peritonitis, and the day has not yet arrived when "surgery must hand back serous tuberculous peritonitis to the internal-medicine clinic with thanks

for the splendid opportunity which a misunderstanding gave to the profession by means of laparotomy, to study tuberculosis in one of the large cavities of the body" (Borchgrevink, Fenger).

CONTRAINDICATIONS TO OPERATION.—In general miliary tuberculosis operation is, of course, contraindicated. In acute localized tuberculosis medicinal hygienic measures should be first employed. Dry fibrous peritonitis presents the greatest difficulties in operation; and as it indicates a stage in the process of healing, surgical measures should not be employed except to meet complications, such as intestinal obstruction. In the ulcerous form with great emaciation and prostration, operation is contraindicated. Lindfors holds that the existence of a pleurisy darkens the prognosis. Active lesions in other parts are contraindications to operation. Temperature is not a contraindication to operation; it specially indicates interference when it appears that the peritoneal tuberculosis is the only active manifestation.

TREATMENT.—General hygienic measures should be instituted in every case of tuberculous peritonitis whether pulmonary complications are present or not and whether operation is done or not. Every attention should be given to feeding and to seeing that the patient gets the maximum amount of fresh air and sunshine. In suitable cases the vaccine therapy should be used.

SURGICAL INDICATIONS.—In tubercular disease anywhere in the human body complete excision of the process where possible is the most positive curative measure. When this cannot be done, excision of the primary focus will frequently relieve the secondary foci. In abdominal tuberculosis complete excision is only exceptionally applicable, as in tubercular inflammation confined to the Cæcum or Fallopian tubes; but as has been shown by Mayo, excision of the primary focus will usually be followed by the complete and permanent healing of the complicating peritoneal inflammation. This primary focus is usually in the Fallopian tubes in the female, and in the appendix or cæcum in the male. Whenever the patient's condition justifies operative interference, the primary focus should be excised, provided it can be located and too much violence does not have to be done to the intestine in breaking up adhesions. Every effort must be made to avoid fecal fistula. In some cases of tubal disease incision with removal of the caseating mass may be substituted satisfactorily for excision. The curative value of

simple incision of the abdomen in suitable cases of tubercular peritonitis has been recognized since 1862, when Sir Spencer Wells first made this discovery. When the primary focus cannot be located or for any other reason cannot be removed, we can hope for fifty per cent. of cures from incision alone. Nothing more than evacuation of fluid and mopping out of the cavity is required. The use of iodine in any form and of other antiseptics and irritants is of doubtful value. Drainage is both useless and dangerous. Adhesions must be handled with the greatest care and should not be interfered with at all except for the evacuation of encysted exudate. Many theories have been advanced in explanation of the good done by simple exploration, the most reasonable being that it results from the hyperæmia of the peritoneum and subperitoneal tissues caused by opening up the abdomen.

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ULCER OF THE STOMACH.

DEFINITION.—A necrobiotic process beginning in the gastric mucosa, consequent upon impaired nutrition and autodigestion, with a tendency to involvement of the muscular and serous coats.

SYNONYMS.—Peptic ulcer, round ulcer, perforating ulcer.

FREQUENCY.—Brinton's original statement, "a peptic ulcer either open or cicatrized is found in 5 per cent. of all autopsies," has been confirmed by Welch, and by Greenough and Joslin, and may be accepted as a correct estimate of its frequency. That it is somewhat more common than formerly may be gathered from the statistics of J. F. Payne, taken at the Mass. General Hospital, of six quinquennial periods, showing the number of cases of gastric and duodenal ulcer respectively 30, 38, 70, 103, 174, 216. These figures, however, may argue only a greater accuracy in diagnosis. That it is more common in northern than in southern climates appears to be a fact. The fact that it is found almost four times as frequently at autopsy in Europe as in this country is accounted for to a great extent by the superior care and skill with which European post-mortems are made.

ETIOLOGY.—Many efforts to assign a specific cause for the simple ulcer have been attempted. These various theories we shall discuss, but let us say at the outset that ulcer of the stomach is a condition to the production of which many factors contribute, and a full recognition of these several causative elements materially assists in the diagnosis.

Gastric ulcer may occur at any age, three days to ninety-six years (Moynihan), but is more frequent in early adult life, eighteen to twenty-eight, and in middle life, thirty-eight to forty-eight (Niles). Welsch's table of 607 cases gives 448 between the ages of twenty and sixty. The average age in women is less than that in men by ten years.

Sex: Brinton's ratio of one male to every two females is approximately correct, though the proportion in females is stated to be as high as five to one. Welch, in an analysis of 1,699 cases,

found ulcer in 40 per cent. of males to 60 per cent. of females. Stawell, in 7,700 post-mortems, found gastric ulcer recorded in 55 men and 41 women. Mayo's latest statistics of gastric ulcer proper give fifty-two men to forty-eight women.

The statistics as to frequency, age, and sex are unreliable because cicatrices, by many observers, are not taken into consideration.

Autodigestion of the mucous membrane of the stomach by the gastric juice plays such an important role in the production of ulcer, that, though not a prime cause, it is well to consider its relation to ulceration first. It is well known that the gastric juice is a corrosive liquid, competent under appropriate conditions to destroy all albuminous substances with which it comes in contact. It is well known that if the individual is in a fairly normal condition, lesions of the mucous membrane heal without delay. These are certainly exposed to the action of the gastric juice. There must be some underlying predisposing cause in peptic ulcer which renders the mucous membrane susceptible to digestion by the gastric juice. We must conclude that there is no one cause of gastric ulcer, but that many conditions, local and constitutional, may lead to its development—the essential factor being the action of the hydrochloric acid on a mucosa of impaired resistance.

Traumatism, external and internal, is an assigned cause of ulcer.

In the various constitutional conditions, hysteria, chlorosis, anemia, post-puerperal states, etc., so frequently associated with ulcer as to be considered etiological factors, there is deficient nutrition. If, in this state, some local lesion of the stomach-wall occurs, by reason of the lowered vitality the gastric juice works its destructive effect.

It is well known that in many septic conditions, typhoid fever, dysentery, tuberculosis, ulcerating surfaces from burns, etc., we have gastric ulcer, usually multiple, appearing. Here the infection is carried by the blood-vessels, leading to thrombosis and cutting off nutrition.

The theory that the fault lies with the central nervous system or with the pneumogastric nerve is somewhat borne out by the results of experimental division of pneumogastric. Turck, of Chicago, has been able to produce ulcers in dogs with unvarying regularity by feeding them cultures of the common colon bacillus.

And, as I have attempted to show, all the conditions, both local and constitutional, impair the nutrition of an area of the stomach; these several conditions may be considered as the prime causes. The pathological process might be arrested at this, were it not that another factor comes into play, namely, the acid gastric juice, which must be considered the essential or active cause in the production of the corroding ulcer of Cruveilhier.

The constant association of an excess of hydrochloric acid with ulcer of the stomach is a clinical observation recognized by all observers from Rokitansky to the present day. Riegel has ascribed to hyperchlorhydria an etiological significance which has been generally accepted. It is well known that gastric ulcer occurs in neurotics—in chlorosis, anemia, hysteria, amenorrhea, etc., and these conditions are associated with hyperacidity. Riegel's views received the endorsement of Ewald, Einhorn, Hemmeter, and others.

This power of the hydrochrolic acid in the gastric juice to destroy the mucous membrane of impaired resistance completes our argument as to the etiology of gastric ulcer.

PATHOLOGY.—**Classification**: There are two distinct forms of ulcer of the stomach, clinically classified as acute and chronic, pathologically as non-indurated and indurated, terms fairly descriptive of the clinical behavior and physical characters of the ulcers. Classified in this way acute ulcer is usually made to cover superficial erosions.

1. **Acute Ulcer**: This small, round or fissured ulcer in 75 per cent. of cases is located in the pyloric zone of the stomach, usually in the neighborhood of the lesser curvature. The edges of the ulcer are invariably soft and free from inflammatory thickening, hence Mayo calls it the non-indurated ulcer. Its base may be formed by the submucous or peritoneal coats. It cannot be detected, unless perforative, from the peritoneal side of the stomach; yet fatal hemorrhage may come from a stomach with a minute lesion. Mayo emphasizes Lund's observation that a sentinel lymphatic gland may be found in the omentum, tributary to the lesion.

2. **Chronic**: Set in a frame of healthy mucous membrane, the chronic ulcer with its rounded contour, sharply-cut, terraced edges, surrounded by a zone of fibrous tissue with puckering of the gastric wall about, funnel-shaped, presents a typical picture.

Its macroscopical appearance defines the pathological process at work: it is a progressive necrobiosis in the course of the obliterated blood-supply.

The surface of the stomach around the ulcer shows chronic inflammation; there is an area of the induration. This varies greatly according to the acuteness or chronicity of the process; venous varicosities appear around the ulcer. In old ulcers the muscular coats of the stomach retract and the mucous membrane flaps like a curtain, concealing the excavation. The floor of the ulcer is always thickened, walls hard, even callous; the peritoneum is dead white or yellow in color and is quite easily distinguished from the pearly luster of the surrounding healthy serosa. As the ulcer erodes its way toward the peritoneal surface, in the majority of cases adhesions are formed about the site of the ulcer to adjacent structures. This is an effort on the part of Nature to prevent perforation; it, however, has the unfortunate effect of anchoring the stomach, inhibiting its natural peristalsis and preventing cicatrization of the ulcer. These adhesions about the stomach, the plastic perigastritis, explain some of the cases of the most difficult digestion we have. Four fifths of all ulcers heal by cicatrization (Fenwick), a restorative process, yet nevertheless responsible for many of the complications that follow in the wake of gastric ulcer. The habitual location of ulcer being the pyloric end of the stomach, the contraction which attends healing of an ulcer in the locality results in stenosis. Ulcer in other parts of the stomach, especially the ulcers that encircle the stomach, result in pouching and deformity of the stomach and in one instance in typical hour-glass contraction. Chronic ulcer, like the acute form, tends toward perforation. Perforation is not always slow in the chronic ulcer; it may occur suddenly without preformed adhesions, yielding symptoms of perforative peritonitis described under acute ulcer. The progress of ulceration toward the serosa may be so slow that adhesions may form before there is penetration of the stomach wall. These adhesions may be sufficient to circumscribe the escaped stomach contents and in this way a localized peritoneal suppuration may form. Fistulous communication may be established with adjacent hollow viscera, a gastrocolic fistula being not unusual. The perigastric adhesions uniting and fixing viscera may give rise to a very decided tumor, confusing to the clinician and misleading to the surgeon at the time

of operation. We do not question but that many of the reported gastrectomies for carcinoma are honest mistakes, the tumor being only a callous ulcer. The relation of ulcer of the stomach to cancer of that organ is a question that has in the last few years received much consideration. It is well known that about forty per cent. of all cancers affect the stomach and that about three per cent. of deaths from all causes are due to gastric cancer. It is further estimated that 6 per cent. of all ulcer cases ultimately have cancer. Christopher Grayham, of Rochester, Minn., participating in the work of the Mayos, makes a very careful study of the clinical relation of ulcer to cancer. His conclusions are:

1. That a great majority of ulcers are located in the pyloric end.

2. That the immediate pylorus comes first, the lesser curvature second.

3. That 50 per cent. or 60 per cent. of patients suffering from carcinoma give three or more years of precancerous history.

4. That a growing per cent. of cancer cases are found with short histories and ulcer demonstrated as the early lesion.

He further concludes that cancer may develop under the same gastric conditions and only manifest this when obstruction or systemic poison makes itself felt.

Number: Fenwick declares that ulcer of the stomach is not a solitary disease. Moynihan reports finding more than one ulcer in 22 cases out of 50. Francine found more than one ulcer in 47 per cent. of autopsies on ulcer cases.

Size: Acute ulcer is usually small, the size of a ten-cent piece or smaller. Chronic ulcer may be as large as the hand, involve an entire surface of the stomach, or even entirely encircle the organ.

Site: Welch's table, assigning the frequency of location of gastric ulcer in the different parts of the stomach, receives the confirmation of autopsical and operative findings. In 793 cases he found the ulcers located as follows:

	Per Cent.
Lesser curvature	36.3
Posterior wall	29.6
Pylorus	12
Anterior wall	8.7
Cardia	6.3
Fundus	3.7
Greater curvature	3.4

75 per cent. of all ulcers in both men and women are situated near the pyloric extremity. The situation influences materially the symptoms, and affects the complications. Mayo claims that the last three quarters of an inch of the pylorus, the canal of Jonnesco, is seldom involved. The usual type is a saddle ulcer on the lesser curvature just above the pylorus.

The progress of the ulcer from the mucous membrane to the serosa may be an exceedingly slow process. As the serosa is invaded adhesions may form to adjacent viscera. The attachment temporarily corks the perforation, but so anchors the walls of the stomach as to prevent closure by cicatrization. The perigastric adhesions circumscribe and confine the infectious matter escaping from the stomach. Unfortunately, perforation may occur without these protective adhesions. We then have developing the most serious complication in the history of ulcer which initiates a new and complicated pathological course with new and urgent symptoms.

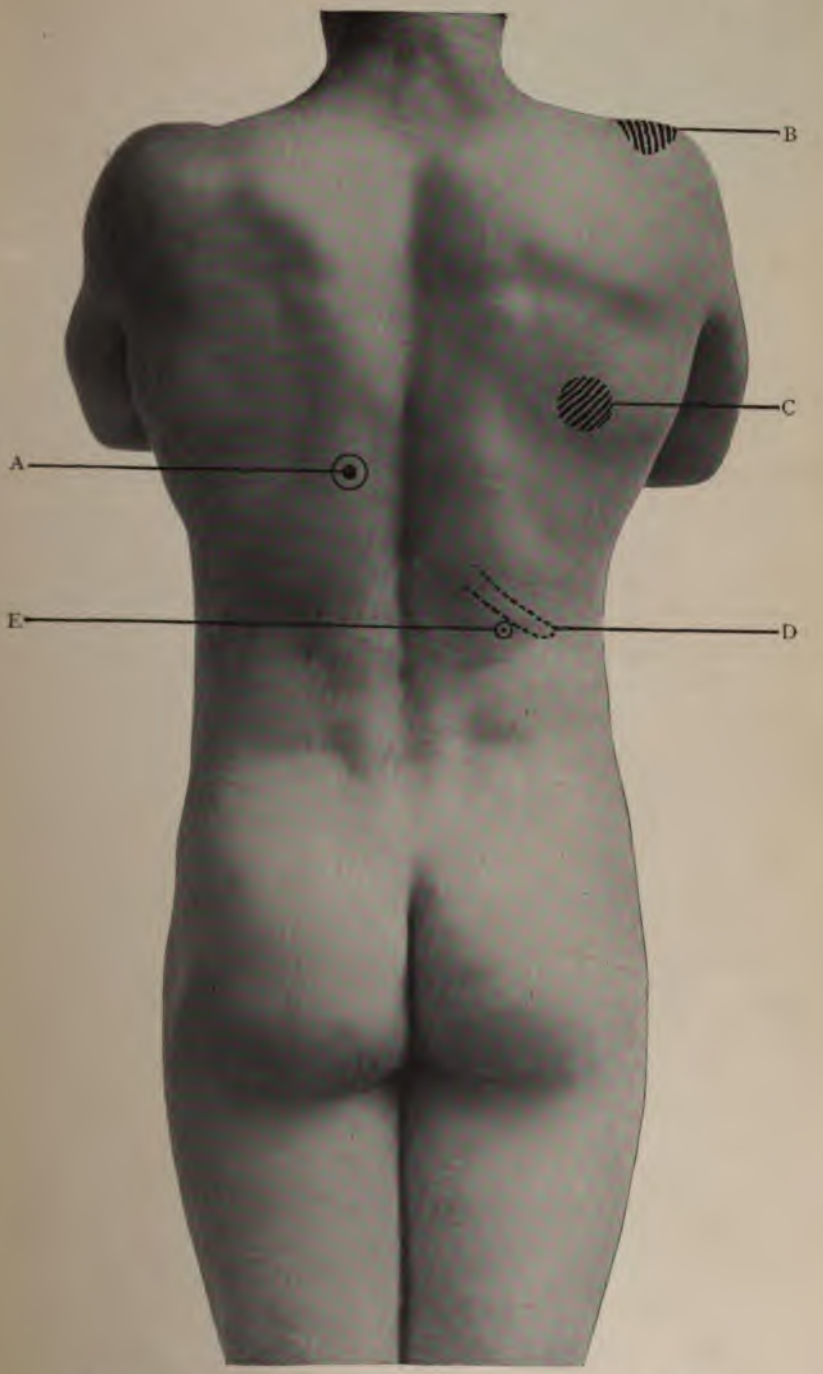
Symptoms.—Ulcer of the stomach presents such a diversified clinical course that any attempt to present a clinical picture, fully covering its symptomatology, would be futile.

Both indurated and non-indurated ulcers may be latent for an indeterminable period and the first evidence of their presence may be some such serious complication as perforation. When recognizable symptoms are present they are pain, tenderness, vomiting, hemorrhage, hyperchlorhydria and evidences of gastric stagnation. All of these symptoms are seldom present in any type of ulcer and not infrequently acute ulcer is manifested by hemorrhage alone. Hematemesis was the first symptom in 75.4 per cent. of one hundred and eighteen cases of acute ulcer of the stomach which were treated in London Hospital by Fenwick.

These general symptoms, while not absolutely diagnostic of ulcer, direct our investigation to that pathology, and we proceed in a systematic way to analyze each symptom.

Pain: In gastric ulcer, actual pain or some modified expression of it is due to three causes: (1) irritation of the sensory nerves by food, (2) irritation from the hyperacid gastric juice, (3) muscular contraction of the walls of the stomach itself. 92.5 per cent. of patients complain of pain (Greenough and Joslin). The distress may appear as a severe gastralgia, described as boring, colicky, or lancinating. The location of the pain is usually in

PLATE I.



SIGNIFICANT POINTS OF PAIN AND TENDERNESS IN DORSAL REGION.

A. Seat of referred pain in gastric ulcer one inch to the left of the tenth to twelfth dorsal vertebræ.
B. Referred hepatic pain over spine of scapula.
C. Referred hepatic pain in back near angle of right scapula.
D. Twelfth rib.
E. Point of renal tenderness in affections of the kidney.

Head's triangle, which lies in the epigastrium to the left of the xiphoid cartilage, between it and the costal cartilage. At least one-half the patients locate the pain in this locality—others in the left hypochondrium. Occasionally the pain may be reflected to the shoulder, a fact which must be borne in mind in differentiating hepatic and biliary troubles. Cardialgia may be severe.

The pain is usually felt immediately after eating; if it is as late as two or three hours, it would point to the ulcer being in the pyloric region, or even in the duodenum. Pain is relieved sometimes by taking food, especially a large quantity of liquid food; this is explained by the dilution of the gastric juice, and also by the presence of neutralizing alkali in the food and saliva. Usually, however, the discomfort excited by ingesta persists until the stomach ejects its contents. Occasionally the pain is relieved by pressure, but, as a rule, even the pressure of moderately-tight clothing is objectionable.

The pain of ulcer is fixed, does not change from place to place. Pain is sometimes felt only at night; this is explained by the posture of the patient and the position of the ulcer. Ulcers of the cardia give rise to little pain. Openchowski's observations on the movements of the stomach are that the stomach-movements extend from a point situated at the junction of the middle and upper third, from this as a fixed point to the pylorus. The cardiac end shows no movement, hence the absence of pain when it is involved.

Tenderness: Epigastric tenderness upon light pressure is one of the most significant signs of ulcer. It is not a point of tenderness, but an area of tenderness as large, perhaps, as the palm of the hand, and it occupies the left epigastric triangle. To elicit this diagnostic sign the touch must be very light; deep pressure may obliterate it.

Tenderness in the dorsal region, recognized long ago by Cruveilhier, and called by him "*point rhachidien,*" a dorsal point of tenderness, is emphasized by Boas as of the greatest diagnostic value. It is almost as constant as the epigastric pain or tenderness, and as it is so sharply circumscribed, for diagnostic purposes it is far more valuable. It is localized at the level and to the left of the tenth to the twelfth dorsal vertebræ. The value of this sign is its constancy, and it does not exist in any other

disease of the stomach or liver. This dorsal tenderness is considered by many as present only in ulcers of the posterior wall of the stomach, but such is not the case; the dorsal tenderness does not depend for its presence upon the site of the ulcer.

Pain and tenderness are certainly the most constant, but not the most characteristic symptoms.

Vomiting: Vomiting occurs at various periods in the history of ulcer; indeed, it may precede the actual ulceration, being a symptom of hyperchlorhydria. In the early stages of ulcer the vomiting is that of irritation. In pyloric ulcer the associate pylorospasm may result in anti-peristalsis. Cicatrization from healed ulcers may result in deformities and constrictions, giving rise to consecutive dilatation with its characteristic vomiting. Vomiting affords such relief to the discomfort that it becomes merely a mechanical and a not altogether objectionable symptom. The vomitus rarely contains bile.

Hemorrhage: Hemorrhage manifests itself as either hematemesis, or melena, or both. The bleeding from an acute ulcer is usually due to the erosion of a small artery. It is therefore sudden and profuse. The capillary oozing, occult bleeding, which attends the destruction of the mucous membrane escapes the patient's observation. If the vomitus or stools are examined after the method of Boas, this early and significant hemorrhage may be detected; however, there may be no symptoms calling for such an examination. Hemorrhage takes place during digestion; usually two hours after taking a full meal. The distention of the stomach has much to do with producing the rupture. The characteristics of hemorrhage from an acute gastric ulcer are emphatically stated by Moynihan to be "spontaneity, abruptness of onset, the rapid loss of a large quantity of blood, the marked tendency to a spontaneous cessation; the infrequency of the repetition of hemorrhage in anything but a trivial quantity and the transience of the resulting anemia." The erosion of the gastric vessel is usually quite painless. The symptoms of acute anemia, vertigo, faintness and weakness are the first to appear. The patient may have a sensation of warmth or trickling of fluid in the stomach. The accumulation of blood in the stomach soon results in vomiting; exceptionally, it may not show until some hours later as melena. The constitutional symptoms are those of internal hemorrhage from other causes, intensified by the

patient's anxiety at the appearance of bright red blood gushing from the mouth. Their severity and duration depend upon the extent of the hemorrhage. Reaction usually prompt. Blood may appear first in the stools; epigastric tenderness and fullness distress the patient. Fatal syncope may follow immediately, as I have twice observed. This termination is quite unusual. All observers agree that such a fatality may be safely prognosticated against. In the physical characteristics of the ulcer we find the explanation for Nature's ability to arrest the hemorrhage. The borders are soft and healthy; the vessels retain their elasticity and retractile power and Nature's hemostatics are operative. With regard to the usual characteristics of hemorrhage from acute ulcer, emphasized by Moynihan, namely, its tendency to recur only in slight quantity and the transience of the resulting anemia, I think the explanation may be found in the withholding of food and the absolute quiet universally observed after a gastric hemorrhage.

In chronic ulcer there is a persistent or recurring loss of a small amount of blood. Even in cases dying from hemorrhage and hemorrhage from the indurated type is the most common cause of death in men who die from gastric ulcer, the bleeding is usually of this character. Occult hemorrhage is constantly present, so much so that its disappearance is considered by many the most reliable evidence of cure. Boas uses both the Guiac and Aloin tests for occult hemorrhage. The Guiac test is the simplest and is said to be very delicate and reliable. 20 c.c. of ether is mixed with 5 c.c. of softened feces. 3 to 5 c.c. of glacial acetic acid are added. The whole is poured into a test tube and extracted again with ether. A few grains of finely powdered Guiac resin are added to the ether extract. The whole is carefully shaken and from 20 to 30 drops of oil of turpentine are added. The whole is shaken again and is then set aside. The color gradually changes to a violet or blue, which is rendered still more intense by the addition of chloroform.

Klunge's Aloin Test.—As much aloin as can be taken up on the tip of a small spatula is shaken up in a test-tube with 3 to 5 c.c. of 60 to 70 per cent. alcohol. The acetic acid and ether extract of the feces as prepared above is treated with 20 to 30 drops of turpentine and then with 10 to 15 drops of the aloin solution. If blood is present the solution assumes a bright red color. If no blood the fluid remains yellow for an hour and then becomes a light pink.

Disturbed Gastric Secretion: To Riegel and his followers is due the credit of establishing the diagnostic significance of the excess of hydrochloric acid in the gastric contents. Ewald, recognizing its value as a symptom, looks upon it as an effect rather than a cause. A very large percentage—90 per cent. according to Hemmeter—show an excess of hydrochloric acid. Under the head of etiology we have clearly stated our conviction that it was the active cause of the ulcer. The constancy of its presence entitles it to a place in the roll of the cardinal symptoms of ulcer. It is undeniably true that many cases of ulcer without an excess of hydrochloric acid are reported; Hemmeter explains this on the very rational grounds of exhaustion of the gastric glands. In acute ulcers, terminating in death from hemorrhage or perforation, hydrochloric acid is absent. May not the destructive course these cases take class them under the head of bacterial necroses? Under certain conditions with favorable soil, etc., the germs are capable of producing the destructive changes without the aid of auto-digestion. I venture to assert that the only reason hyperacidity has not received an important rank in the eyes of clinicians is the rather tedious test required for its detection and quantitative analysis. The simplest tests are the following:

Test-Meal: The best test-meal is the test-breakfast of Ewald and Boas. It consists of an ordinary dry roll and a definite quantity of warm water (10 fl. oz.). This should be withdrawn within one hour. The method of expression should suffice to obtain the stomach contents. By having the patient bear down as if at stool, the increased intra-abdominal pressure should cause the chyme to rise in the tube. If this fails we may use a Boas bulb or an ordinary aspirating bottle with a low vacuum to start the syphonage.

Chemical Tests of the Stomach-Contents: There are a number of chemical tests to be employed, all open to the criticism of being rather tedious to the general practitioner, yet in hospital work the investigation can be easily carried out.

(*a*) *Test for the Presence of Free Acids:* This is done with Congo Red, which occurs as a fine reddish-brown powder. This may be dissolved in water and a portion of the sample added directly, or a test-paper may be made by soaking filter-paper in the solution for several hours and then drying it. In either case,

the red of the dye will be changed to a bright blue in the presence of free acid.

(*b*) *Test for Free HCl:* Probably the simplest and most reliable test is the phloroglucin-vanillin test. Günzberg's formula is:

Phloroglucin	grm. ii (gr. xxx)
Vanillin	grm. i (gr. xv)
Absolute alcohol	c.c. xxx (fl. oz. 1)

Boas modifies this, substituting for the 30 c.c. of absolute alcohol 100 c.c. of 80 per cent. alcohol, which he claims is more stable; but both readily undergo decomposition, and should be kept in a dark-colored, well-stoppered bottle. This solution is pale-yellow in color. To use, place a few drops of this reagent in a porcelain dish, add an equal amount of the gastric contents, and very gently heat over a flame, or better on a water bath, so that the fluid does not boil but evaporates slowly. If free HCl is present, a fine rose-tint will develop at the edge where the mixture has dried, and blowing on the dish will cause rose-colored stripes to appear. The greater the proportion of acid, the more pronounced will the reddish tinge be.

Stagnation of gastric contents frequently results from active indurative ulcer of the pylorus, from infiltration and thickening of the stomach wall, partial cicatrization with contraction, from inflammatory shortening of the gastrohepatic omentum, from perigastric adhesions, etc. Hypertrophy of the muscle of the stomach wall and possibly pyloric spasm are also causes. Interference with stomach motility as shown by the retention of food beyond the proper time of stomach digestion is a symptom of great diagnostic value and also has an important bearing on the question of surgical treatment.

Other tests that are recommended are Töpfer's or the dimethyl-amido-azobenzol, and Boas's or the resorcin test. There are many others, but the three mentioned are the most reliable.

(*c*) *Quantitative Test for Free and Combined HCl:* Töpfer's method.—Into each of three beakers (A, B, and C) measure with a pipette 5 c.c. of gastric juice. Titrate each of them with decinormal NaOH (4 grms. to the liter), using as indicators, in A phenolphthalein, and adding the alkali until the liquid is a faint red; in B three or four drops of a 1 per cent. solution of alizarin

sodium sulphonate in water. The NaOH must be added until the liquid is a pure violet color, not stopping with a reddish-violet shade. The exact shade is very nearly that given by the indicator to a 1 per cent. solution of the common sodium phosphate. With C the indicator is three or four drops of a five-tenths per cent. alcoholic solution of dimethyl-amido-azobenzol. HCl gives a red color with this, and the NaOH is added until this disappears and the color changes to yellow.

The phenolphthalein is turned red by all the compounds with an acid reaction; the alizarin sulphonate reacts with all but the acid albumin; and the dimethyl-amido-azobenzol only with the free HCl. The test of A gives the total acidity; of C the free, or uncombined HCl; and the difference between A and B the loosely combined HCl, or that which is in combination with the albuminous compounds. The difference between A and B–C represents the acidity due to the phosphates and organic acids.

Calculate from the amounts of NaOH used the percentages of each of these, remembering that one c.c. of the alkali contains 0.004 grm. and neutralizes 0.00364 grm. of HCl. The amounts of acid phosphates and organic acids must be expressed as equal to a certain amount of HCl.

The age, sex, appearance, and history of the patient must always be taken into consideration in summing up the diagnosis of ulcer. The cardinal or characteristic symptoms, pain, local tenderness, vomiting, hematemesis, altered gastric secretion, are in themselves sufficient to make a positive diagnosis. If with these we couple anemia, hysteria, or chlorosis, in a pale, rather languid individual, of constipated habit, a sufferer from heart-burn—a chronic dyspeptic—we have an array of symptoms which typifies ulcer of the stomach.

The fulminating type of gastric ulcer, resulting quickly in perforation or hemorrhage, is more likely to appear in young individuals; and, as previously stated, bacterial necrobiosis is no doubt responsible for the rapidly destructive course.

The symptoms of pyloric obstruction, due to cicatrization and contraction, usually develop after a clear history of gastric ulcer located in the region of the pylorus. Here the presence of a tumor, due to the complicating paragastritis and enlargement of the neighboring glands, while not a usual feature of gastric

ulcer, occurs sufficiently often to be taken into consideration in the differential diagnosis.

Pain, tenderness, vomiting, hemorrhage, important as they are, may one and all be absent. Indeed, the most serious form of gastric ulcer is the latent type, which suddenly discloses its existence by perforation or fatal hemorrhage. This form of ulcer usually occupies the anterior wall of the stomach, occurs in individuals of rather advanced age, and is small in area. The absence of local distress and associate symptoms throws nature off her guard and exposes the patient to imprudence in diet and exercise, which may precipitate fatal complication.

COMPLICATIONS.—(a) **Perforation**: This most frequent and dangerous of all complications of ulcer of the stomach deserves lengthy consideration. The frequency of occurrence of perforation is given as 6 per cent. to 15 per cent.; the latter figure will perhaps be found to be too low after another year's work. A curious fact is that, while gastric ulcer occupies most frequently the posterior wall, perforation is far more common in the anterior wall. At least 80 per cent. of all cases of perforation have their situation on the anterior surface. Of 31 cases of perforated gastric ulcers submitted to operation, Barling found that in 22 perforation was on the anterior surface. This is fortunate, for although the danger of peritonitis is enhanced, the chances for quick and thorough treatment are better.

The direction of perforation depends upon the site of the ulcer; if on the anterior surface, greater curvature, or at the pylorus, without acquired adhesions, the opening is into the general peritoneal cavity. If the ulcer occupies the posterior wall, adhesions are likely to form to the pancreas, which may protect against perforation; should perforation occur, it communicates with the lesser peritoneal cavity.

The progress of ulceration toward the serosa may be so slow that adhesions may be formed before there is penetration of the stomach-wall. These adhesions may be sufficient to circumscribe or sacculate the escaped stomach-contents, and in this way a localized peritoneal suppuration may form. Even after perforation of the stomach, in a few cases (six are reported) adventitious attachments are sufficient to bring about healing and restoration to health. Fistulous communications may be

established with adjacent intestines. Septic infection leading to abscess of the liver, spleen, and parastomachal glands is a not unheard of complication of gastric ulceration.

But, unfortunately, perforation is generally a sudden complication, and the contents of the stomach are poured into the peritoneal cavity, there to give rise to acute general peritonitis.

Perforation is sometimes attended by the erosion of a large artery, which materially complicates the symptomatology, and hastens the death of the patient.

Perforation occurs more frequently in men of advanced age and in young women; and while it occurs without any assignable cause other than the ulceration, yet in most instances the accident happens when the stomach is full, or during vomiting, or after some unusual exertion. Hemmeter's reasons for the frequency of the perforation of the anterior wall, previously given, are very satisfactory.

Impending perforation cannot, as a rule, be recognized. It is true that in a few rare instances the localized peritoneal irritation may warn us of the approaching danger; this is only exceptionally true. The actual perforation declares itself by symptoms not always characteristic of gastric perforation, yet impressively denotive of some serious intra-abdominal complication.

Symptoms of Acute Perforation: The occurrence of sudden perforation is attended by the general symptoms of abdominal shock or peritonism, the most characteristic of which is an acute, sharp, lancinating pain in the upper part of the abdomen, frequently confined to the epigastrium, radiating through to the back, and not infrequently to the interscapular region. The patient and surgeon recognize that it is something out of the ordinary. Shock, usually profound, quickly supervenes. The intensity of the suffering and the sudden invasion of the peritoneum explain this phenomenon. The pulse becomes rapid and feeble, the skin is cold and bathed in a colliquative sweat, respiration is hurried and thoracic, the facial expression denotes great anxiety, the temperature is normal or subnormal. Syncope may attend the sudden onset of the pain. Vomiting is absent in 25 to 40 per cent. of cases. Struve, who investigated 42 cases on this point, says that vomiting is absent only when the perforation is large. The

vomitus rarely contains blood; in perforation blood is absent from the vomitus in 80 per cent. of cases (Robson). The abdominal muscles are rigid and retracted; this is a most important and diagnostic sign, and, if coupled with pain and marked local tenderness, warrants surgical intervention. To it Deaver attaches the greatest importance. There may be general abdominal hyperesthesia, but special localized or point-tenderness is a symptom equal in value to McBurney's point in appendicitis. Should hemorrhage occur at the time of perforation, the above symptoms would be somewhat modified, the rigidity perhaps less marked, but the shock more profound and syncope of a recurring type.

In a few hours the immediate symptoms, as above given, which are due to the actual occurrence of perforation, yield to the symptoms of its effect. Slowly reaction takes place, the pulse becomes stronger but remains fast, the surface and body temperature rise, the pain subsides, but soreness and tenderness persist. Rigidity is displaced by progressive tympany. We now note the disappearance of normal hepatic or splenic dulness; this sign cannot be employed, and really does not exist, until there is some relaxation of the primary rigidity of the muscles.

Peritonitis now supervenes, marked by rising temperature usually, though the temperature may remain low; a fast pulse, with tendency to grow weaker and faster; the patient assumes the characteristic, protective dorsal position; the pallor of the initial shock has now changed to a rather dusky hue; vomiting recurs; the bowels are constipated and resist efforts to move them; the belly distention increases, and palpation and percussion may reveal rapidly accumulating fluid. If the patient does not die of shock in the first few hours, death from general peritonitis in a short time is almost inevitable.

In the diagnosis of perforation from gastric ulcer, too much stress cannot be laid upon the history, immediate and remote. Pain alone, sudden and severe, when supported by a clear history of gastric ulcer, warrants surgical intervention. Perforation may not be found, yet conditions justifying the undertaking will be. History of ulcer, pain, and rigidity, with or without shock, demand interference. Valuable to the diagnosis as the history may be, yet the possibility of latent ulcer must always be borne in mind, and surgical means employed if the other symptoms war-

rant it. As some one has aptly said, "A timely diagnosis presupposes a timely operation." Unless the patient is actually dying of shock, operative measures should be resorted to immediately upon the recognition of the perforation. The mortality after operation, as given by Finney, is now 48 per cent. Is it not safe to predict that in the near future it will be reduced to less than one-half this figure?

Differential Diagnosis of Perforation: *Acute phlegmonous cholecystitis* may be mistaken for perforation of a latent gastric ulcer. In both instances in this dilemma there may be an entire absence of a guiding clinical history. The pain may be epigastric in both. In ulcer it is usually a little to the left in the epigastric or hypochondriac region, often felt acutely, close to the scapula of the same side. In cholecystitis the pain is generally over the gall-bladder, is referred upward to the right thorax and shoulder, and quite commonly to the right iliac fossa. In perforated ulcer the tenderness varies somewhat with the site of the ulcer; usually it is most acute in the left epigastric triangle, but may be much lower down along the costal margins. In cholecystitis there is exquisite tenderness over the gall-bladder. In perforated ulcer there is shock; and low temperature obtains until peritonitis supervenes. In cholecystitis high temperature frequently marks the initiation. Vomiting of blood is so frequently absent in perforation that its absence signifies nothing; its presence is conclusive. In ulcer there is no tumor, while in cholecystitis there may be.

Acute Pancreatitis: The shock which marks the onset of this form of pancreatitis suggests visceral perforation. Unless a perforated ulcer of the stomach is attended by free bleeding, the shock is less profound, and reaction generally follows promptly; in pancreatitis patients frequently succumb to the initial shock. In less vicious cases the symptoms are very confusing. The tenderness in perforated ulcer is superficial, and there is general hyperesthesia of the abdomen; in pancreatitis the tenderness is deep-seated. In perforation the stomach is usually collapsed; in pancreatitis it may be distended. In perforation tympany quickly develops, with effacement of normal areas of dulness; in pancreatitis there may also be tympany, but it does not obscure hepatic and splenic dulness.

Intestinal Perforation: In intestinal perforation we are usually guided by a history of typhoid fever or of some local lesion. The pain is, as a rule, at the umbilicus or in the iliac region. The evolving peritonitis at first is subumbilical, and then becomes general. And there is the absence of the superficial tenderness and characteristically referred pains.

Ectopic Gestation: In a case of ectopic gestation pregnancy was not suspected, and the gastric disturbance was attributed to indigestion. Upon rupture the pain was felt entirely in the epigastric region, and the case closely resembled perforated gastric ulcer. The acute anemia, recurring syncope, failing pulse, and the detection of free blood in the peritoneal cavity, and a tumor in the pelvis, guided me to a correct diagnosis.

Appendicitis has been a source of error. Careful application of physical signs should lead one to discriminate between the two conditions.

Prognosis of Perforation: Finney gives an operative mortality after perforation of 48 per cent.; others place it as high as 62 per cent.; but evidently with improved diagnosis these figures are bound to be entirely too high. Keen's latest operative mortality is given as 17 per cent. The prognosis depends upon the site of the ulcer, the quantity and character of the stomach-contents, but above all upon the time that elapses between the perforation and operation.

(*b*) **Subphrenic Abscess (Chronic Perforation):** After acquiring adhesions, should an ulcer on the anterior surface of the stomach perforate, the pus might be so circumscribed as to be confined in one pocket or area of the general peritoneal cavity; usually such collections or pockets of pus are to the right side of the falciform ligament of the liver, and may push the liver down to a considerable extent. This is an intra-peritoneal abscess, but subphrenic in character.

In perforation of the posterior wall of the stomach, cases of true subphrenic abscess, such as were first described by Barlow, undoubtedly occur. Leyden, who developed the diagnosis of the condition, calls it a "pyopneumothorax subphrenicus." This name is scarcely applicable, since gas is not necessarily present and causes a variance in the physical signs. In perforation of the stomach, as a rule, there is gas as well as pus; the gas, how-

ever, does not necessarily come from the stomach, but may be due to the gas-producing bacillus.

The pathology and symptomatology of subphrenic abscess are in their developmental stage, and no doubt this complication of gastric ulcer is often unrecognized. It is of such vast importance that we reserve it for individual consideration.

Hour-glass Contraction: In hour-glass contraction the pathological process is either cicatrization or peritoneal adhesions. Cicatrized ulcer of the stomach is the preponderating cause of this deformity. As a rule ulcer resulting in contraction is situated near the middle of the stomach and is a gradual process.

The general symptomatology rests upon the detection of a small or contracted stomach, a condition that is rarely met with except in cancer in the aged; and the diagnosis is reached largely by eliminating cancer. It is true that instances are reported in which persons have lived to middle life in good health, and autopsy has shown congenital constriction. While such a quiescent course may be true of congenital deformity, acquired constrictions usually present a very definite history and gastric symptoms pronounced, but not specifically diagnostic. Indeed, Sievers says the diagnosis is exceedingly difficult, and that the condition is often accidentally discovered at necropsy.

The symptoms are those of great discomfort after eating, which is followed by vomiting of a distressing form. The vomitus is usually small in quantity, and does not relieve the pain which persists more or less all the time. There may be, though rarely, hemorrhage if there is coexisting ulceration. Emaciation is rapid. Wölfler's sign, which he regards as pathognomonic, is: if we inject fluid into an hour-glass stomach, it does not all return through the tube, but seems to disappear as through a larger opening. He further observed that after lavage, when the stomach appeared to be clean, there would suddenly return a quantity of undigested food. Riegel has proved that the presence of food after lavage does not mean hour-glass constriction; this phenomenon may occur in simple dilatation. Probably the most reliable signs are the presence of succussion after we have emptied the stomach through a tube, and a study of the outlines of the stomach by auscultatory percussion after distention with fluid or gas. In emaciated individuals, when the stomach is distended, palpation may show the constricting notch.

The course and prognosis of hour-glass constriction is practically the same as that of benign pyloric stenosis; the fundus or cardiac compartment becomes dilated, its motor power impaired, and death finally ensues from insufficient nourishment.

The diagnosis rests upon the history of ulcer, or perigastric inflammation.

(e) **Pyloric Stenosis**: The location of ulcer habitually near the pylorus leads to one of the most serious and distressing complications in its history. In acute ulcer the associate hyperacidity, by its irritation of the exposed nerves, excites pylorospasm of such persistent character as to lead to hypertrophy and obstruction. Organic constriction from cicatricial contraction and paragastritis, after perforation, is the most frequent surgical complication in the history of ulcer. For its consideration see Pyloric Obstruction.

DIFFERENTIAL DIAGNOSIS.—**Carcinoma of the Stomach**: An ulcer of the stomach may have a varying history through a period of years; in carcinoma the symptoms intensify from the beginning to the termination, which is rarely longer than eighteen months. Ulcer usually causes pain; cancer may not until obstructive symptoms appear. In ulcer vomiting relieves pain; in cancer it does not. In ulcer the appetite is unimpaired; in cancer anorexia is marked. Cachexia is more marked in the malignant trouble. In ulcer there may be dilatation of the stomach, but without a permanent tumor; in carcinoma a tumor is generally found, preceding the development of dilatation. Only in the late stages of ulcer from cicatricial contraction of the pylorus is the vomiting that of dilatation of the stomach; in cancer there is early pyloric obstruction and the consequent vomiting of retention. The vomitus from ulceration may not show blood; in carcinoma blood is frequently present in the vomitus, though in small quantity. The stomach-contents in ulcer shows excess of hydrochloric acid and absence of lactic acid; in carcinoma the reverse is the case. In the late stages of carcinoma there are metastases, edema of the extremities, urticaria and insomnia, with rapid decline.

Gastralgia: Neurotic females especially suffer from pain and vomiting suggestive of ulcer. This gastric neurosis is even marked by severe hemorrhage at times. It is characterized by severe pain

which, however, usually comes and goes spontaneously and bears no relation to the taking of food. It is inconstant in its location and is not attended by tenderness. It does not necessarily impair the general health. Moynihan lays great stress on the variability of the symptoms and the utter lack of order in their occurrence in making a diagnosis of this condition.

Gastritis may exist for years with periodic exacerbations. There is no special point of tenderness, vomiting is not always present, and if so, is attributable to gross errors in diet or to alcoholic excesses. There may be hyperchlorhydria, but not necessarily so. There is no progressive anemia, as hemorrhage is very rare. These patients are often high-livers and obese.

Duodenal Ulcer:

Gastric Ulcer.	*Duodenal Ulcer.*
Age.—Most commonly under forty years.	Forty years or after.
Sex. — More frequent in young women.	In men eight to one.
Vomiting.—A constant symptom.	Not conspicuous, frequently absent.
Time of Vomiting.—Promptly after eating, rarely longer than one hour.	Two to four hours after taking food.
Hematemesis. — Frequently present, in almost every case at some time in its history.	Infrequent.
Pain.—In the epigastrium to the left of the median line, and in the left hypochondrium.	In the right hypochondrium or parasternal line.
Time of Pain.—Usually, immediately after eating.	Two to four hours after eating.
Radiation of Pain.—To the back, and to the left shoulder.	Usually downward, occasionally to the chest.
Tenderness.—Left epigastric triangle, and point-tenderness in the back to the left of the ninth to twelfth thoracic vertebræ.	Over the duodenum, near the region of the gall-bladder, diffused along the right costal arch.
Melena.—Only occasionally seen.	Quite common.
Jaundice.—Occurs only as a complication.	Though an occasional, yet a valuable symptom.

PROGNOSIS.—Mansell Moullin's investigations are the most recent and instructive as to the prognosis of ulcer. There were in the London Hospital within the period from 1897 to 1902 exactly five hundred gastric ulcers; 98 of the patients were men, 402 women. 48, or nearly 10 per cent., died from perforation; 2.5 per cent. from hemorrhage, 5.5 per cent. from other causes; total number of deaths 89, or 18 per cent. Of the 500 patients, 214, or 42 per cent., had been in the hospital before and relapsed. Some authors give the mortality of gastric ulcer treated medically as 50 per cent. In seeking to arrive at the correct prognosis of gastric ulcer, we must make the distinction between patients who die *with* and those that die *from* gastric ulcer. We have already alluded to the frequent association of cardiac, renal and pulmonary disease with ulcer of the stomach. In 100 cases showing the cause of death when an open ulcer was found, 74 per cent., at autopsy, showed that ulcer or its effects were immediately responsible for death; 26 of the cases succumbed to some intercurrent disease (Fenwick).

TREATMENT.—The dividing line between the medical and surgical aspects of gastric ulcer is not definitely settled. The surgical treatment is the growth of only a few years and while the surgical nature of some phases of the disease has been determined, for others, time is yet required for operative experience and post-operative observation before their status can be fixed. The best surgical as well as medical opinion classes gastric ulcer per se as a medical disease. In any ulcer announcing its presence for the first time by recognizable symptoms, and in which perforation has not taken place, medical treatment should be given a careful trial. To set an arbitrary time limit for this is difficult, in fact, impossible. The possible surgical nature of the case should be kept in mind from the start, complications definitely known to be surgical should be carefully watched for, and just so soon as it can be seen that the patient is losing ground, or is even at a standstill, the question of surgical intervention should be considered. In this event what are the operative procedures that may be instituted and what can be accomplished by each?

Excision: Excision or in suitable cases inversion of the ulcer or ulcers present is definitely and permanently a curative measure. Anything short of this in the way of a surgical procedure is to a certain extent a makeshift and may without prejudice be considered

rather palliative than curative. Non-indurated ulcer with those rare exceptions of mucous erosions involving apparently the entire mucous membrane of the organ, are most suited physically for this plan of treatment, but with the means at our command, it is often impossible to locate them. The indurated ulcer, which can usually be readily found by reason of the induration, may be too large and the surrounding tissue too stiff for inversion. Excision often becomes a serious and rarely an impossible procedure, by reason of adhesions present or even on account of the extent of stomach involvement. Indeed at times the excision would necessitate a partial gastrectomy that would be little short of complete excision of the organ.

Pylorectomy: This operation as recommended by Rodman is a method of excision that removes the so-called ulcer-bearing area and has the advantage of not only removing active and cicatrized ulcers, but provides to a great extent against future ulceration, benign or malignant, by removing that portion of the stomach most often affected. The objections to it are that in the hands of the ordinary surgeon it is quite a serious operation with a very definite mortality, and it will at times necessarily fail to reach unsuspected non-indurated ulcers, and even recognized indurated ulcers lying beyond the limits to which it can be carried. Then, too, gastrojejunostomy must be done in addition to reëstablish the continuity of the intestinal canal.

Gastroenterostomy: Gastroenterostomy, or, more accurately, gastrojejunostomy, is in itself only a palliative measure, but one that has been practically of great value in the treatment of gastric ulcer. It is essentially a drainage operation and when done by the posterior, no loop, suture method, it fulfills this condition perfectly. It will relieve all symptoms resulting directly or indirectly from actual pyloric obstruction, inflammatory, cicatricial or spasmodic, if indeed there is such a thing as the last. It relieves stagnation, fermentation and consequent distension of the stomach. By setting the stomach muscle at rest, checking hyperchlorhydria, and preventing irritation of the ulcer by the passage of food over it, it creates a favorable condition for spontaneous healing. Its deficiencies are: First of all, it is a radical change from Nature's method of communication between the stomach and bowel. (This objection is largely sentimental apparently.) Secondly, it does not remove the

ulcer; next, it has little or no influence over ulcers not situated at the pylorus, and in pyloric ulcer it can do little beyond relieving symptoms directly or indirectly due to obstruction. An ulcer at the pylorus has no better reason for actual healing after gastrojejunostomy than an ulcer situated, say, on the lesser curvature some distance from it.

Pyloroplasty: This operation is obsolete, but a modification of it after Finney's method, which is, strictly speaking, a gastroduodenostomy, has some theoretical advantage in that the natural opening from the stomach into the duodenum is preserved in its function, while actual obstruction is relieved by enlarging the opening. It has several disadvantages, a serious one being the difficulty of the operation. The work is done on the diseased portion of the stomach, frequently much distorted by induration, cicatrization or adhesions. It can have no possible influence on ulcers situated elsewhere than at the pyloric opening. Like gastrojejunostomy, it is a palliative drainage operation; in fact, it meets much the same indications and does not meet them as well.

Jejunostomy: In contrast with the operations already considered, jejunostomy has nothing to recommend it except the ease and rapidity with which it can be done. When the anatomy of the stomach distorted by pathologic changes does not lend itself to more satisfactory measures jejunostomy may be done to prevent starvation.

So long as palliative operations are the operations of choice in the treatment of gastric ulcer, even with the most careful selection of cases and the elimination of all neurasthenics, we will have a certain per cent. of failures to get either anatomic or symptomatic cures. Conditions must be met as they arise in individual cases and it is impossible to establish arbitrary rules of operative procedure to meet the exigencies, anatomic, pathologic, and otherwise of all cases, but it is safe to say that the ideal surgical treatment of gastric ulcer is excision, and this should usually take the form of pylorectomy. To make this operation generally applicable greater technical skill in operating is required and some more satisfactory method of locating ulcers, particularly the non-indurative variety.

A consideration of the surgery of gastric ulcer could not in any

sense be satisfactory without special attention to the surgery of perforation and of hemorrhage.

Acute Perforation: The necessity for early surgical interference in acute perforation is so apparent as to admit of no discussion. The abdomen must be opened, the perforation occluded, a toilet made of the peritoneum, and, if possible, provision made for the permanent relief of the stomach. Whenever possible, the opening should be closed by suture. If for any reason, such as the size of the opening, friability of the tissue, extent of induration, etc., this cannot be done, an effort should be made to plug the opening with omentum. Failing this, the stomach might be attached to the parietal wound, making the opening extra-peritoneal, but usually it will be necessary by gauze tamponade to provide against extravasation until the cavity has been protected by adhesions. Lavage of the peritoneal cavity will at times be demanded to get rid of extravasated stomach contents. Exceptionally, sponging of a more or less extended area is all that will be required.

As to the stomach itself, kinking of the pylorus as a result of the closure of the perforation may necessitate gastrojejunostomy. The presence of other ulcers may invite some radical or palliative procedure for their relief, but here we are operating to save life and the risk of prolonged operative procedure is too serious for us to lose time in attempting to do complete surgery. Here is a condition that calls for the highest surgical judgment on the part of the operator and each case must be handled strictly upon its own method.

Chronic Perforation: We may never have symptoms from this type of perforation, as the cavity is frequently protected by preformed adhesions. When symptoms do develop they result from the formation of subphrenic abscess which must be handled on general surgical principles and with small chance of doing direct stomach surgery.

Hemorrhage: Hemorrhage in indurated ulcer is usually the result of distension of the stomach consequent upon pyloric obstruction, according to Moynihan. In most instances it is promptly relieved by drainage of the organ. Exceptionally, we find that this is not true in the indurated ulcer and in the non-indurated type the exceptions are frequent indeed. Rodman states that hemorrhage in acute ulcer yields promptly to medical treatment (he recommends

lavage at 120 to 130 degrees Fahrenheit for severe cases) and adds that surgical intervention is wholly unnecessary. Like all other dogmatic generalizations in medicine this is wrong. Lives have undoubtedly been lost from hemorrhage in acute ulcers that might have been saved by operation. A more conservative statement would be that the extreme rarity of death from hemorrhage in acute ulcer, the unsatisfactory results from drainage operation for its relief, and the extreme difficulty at present of locating the bleeding point or points makes surgical intervention of very doubtful propriety. The frequent failure in acute ulcer and the occasional failure in chronic ulcer of drainage operations to relieve hemorrhage but emphasizes the position already taken that the ideal surgical treatment of gastric ulcer is excision.

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CARCINOMA OF THE STOMACH.

DEFINITION.—An atypical epithelial neoplasm, primarily affecting the glandular structure of the stomach.

Frequency.—About one-fourth of all carcinomata affect the stomach. Next to the uterus, cancer of the stomach is the most frequent form of cancerous disease. In an analysis of 30,000 cases of cancer Welch found the stomach involved in 21.4 per cent. Cancer of the stomach, as of all organs, is undoubtedly increasing in frequency.

ETIOLOGY.—We are as yet in total ignorance concerning the etiology of cancer. This statement is made with the full knowledge of recent bacteriological work, from which much may be hoped; but nothing conclusive has as yet developed.

Heredity: Ancestral history will show heredity in 6 per cent. to 9 per cent. of cases. Less importance is ascribed to this influence by investigators than formerly, yet life insurance companies, figuring very closely upon its bearing in vital risk, increase the premium where the tendency seems marked.

Age: While writers of a generation ago held that gastric cancer could be left out of consideration in the diagnosis of obscure cases if the patient was young, yet later writers recognize the occurrence of a certain percentage of cases in early periods of life (Osler and McCrae). Cheney, in analyzing 150 cases of cancer of the stomach, found that 20 occurred in individuals under 30 years of age. Six cases are reported in children under 10 years of age, and 13 cases between ten and twenty years. While admitting its occurrence in the young as a possibility, it remains a guiding clinical fact that about 75 per cent. of all cases of cancer of the stomach occur between the ages of 40 and 70 years.

Sex: Welch's table shows the relative frequency of the disease as 1,233 men to 987 women; other observers record even a greater relative frequency in males.

There are certain factors which, acting as irritants, are considered to have a predisposing influence; such are gastric ulcer,

chronic gastritis, and traumatism. 70 per cent. of the cases observed by Graham gave a precancerous history. Mayo states that 54 per cent. of his cases have their origin in ulcer of the stomach.

PATHOLOGY.—Cancer of the stomach is overwhelmingly primary. Ewald considers secondary involvement of the stomach by cancer so rare as to be almost a pathological curiosity, yet Welch reports 37 cases, and Osler adds 3 of secondary gastric cancer.

Classification: (1) *Medullary,* (2) *Cylindrical-celled,* (3) *Scirrhous,* (4) *Colloid,* are the four histological varieties of cancer. The surgeon should be familiar with the macroscopical appearance of these several forms, as they have a definite bearing upon the limitations of surgery, and are of prognostic import.

1. *Medullary* cancer shows one tumor with elevated edges, and central ulceration and depression. It is ragged in outline. The bowl-shaped central ulceration leads to involvement of the peritoneum (Hemmeter).

2. The *cylindrical-celled* or *adeno-carcinoma* is a proliferating tumor forming distinct prominences. It gives rise to frequent hemorrhage, and shows a tendency to slough; large portions may be cast off in this way and be found in the vomitus.

3. *Scirrhous:* The distinguishing characteristic of this form of cancer gives it its name, the hard cancer. It shows a flat cicatrix-like appearance with corroded surface, and shades off into healthy mucosa. It is usually at the pylorus, and is often overlooked, secondary growths of other organs engrossing attention to the exclusion of the primary trouble. The tissues of the pylorus becoming infiltrated, a ring-like circular induration results, and the connective-tissue stroma contracts the lumen of the pylorus until finally it becomes impermeable. In the other forms we find extensive infiltration with no contraction (Senn).

4. The *colloid* growth is a gelatinous, translucent thickening of the stomach-wall. "They present a slimy, mucus-like feel" (Hemmeter).

Seat: The most frequent seat of cancer is 2 to 4 cm. from the pyloric extremity, on or near the lesser curvature. From this point it spreads most rapidly in the submucosa toward the cardia. Its growth toward the duodenal side is slow.

Extension of cancer is by contiguity, chiefly in the submucosal tissue, and by the blood, as through the portal veins to the liver, but chiefly by lymphatics. Secondary deposits appear in the lymphatic glands, liver, peritoneum, pancréas, etc. Cunéo says that invasion of the duodenum rarely results from cancer of the pylorus. He emphasizes the early involvement of the parastomachal ganglia and the necessity of their removal in operation. The lymphatics of the lesser curvature lie in the stomach wall and involvement of its entire length can be suspected in every case of pyloric cancer. The lymph channels and nodes upon the greater curvature are comparatively few in number, draining approximately one third only of the adjacent stomach. The lymph nodes here lie in the great omentum and the lymph current is from left to right. It has been demonstrated practically that the fundus and two thirds of the greater curvature of the stomach escape infection in pyloric cancer.

Stomach-Changes: In carcinomatous infiltration the stomach-wall will show general concentric contraction, so that the stomach may be converted into a tube which is merely the continuation of the esophagus. When the cardia is the seat of cancer, atrophy of the stomach follows with dilatation of the esophagus. In all forms obstructing the pyloric extremity, dilatation and displacement inevitably occur. Peritoneal adhesions produce deformities and displacements of the stomach.

Blood-Changes: The most usual alteration of the blood in cancer of the stomach is a decrease in the number of red corpuscles, some increase in the white, and the presence of nucleated red blood-corpuscles. If the blood-count goes below one million, say Osler and McCrae, it points to pernicious anemia and not to cancer.

The extent of the local involvement may not be indicated by the general symptoms.

Superficial ulceration occurs to a variable extent in all forms of cancer. Deep ulceration leading to perforation is comparatively infrequent. As the process is chronic, serous adhesions are usually acquired, and perforation rarely takes place into the general peritoneal cavity; yet a case is reported of death from perforation-peritonitis, in which autopsy revealed the presence of an unsuspected gastric cancer.

SYMPTOMS.—The symptomatology of gastric cancer presents a very diversified picture, nor do the symptoms conform to the pathological findings.

The character and location of the neoplasm, and the age of the patient materially influence the manifestations of the disease. Every gradation from the most active and pronounced symptom to the latent or unsuspected growth is met with. Usually, however, there is a complexus of symptoms fairly constant in their presence, typifying gastric malignancy. Frequently gastric cancer develops as secondary to chronic gastritis or gastric ulcer; in such cases the patient for a long time has had his attention fixed upon his dyspeptic symptoms. Anorexia, repugnance to certain articles of food, especially meats, general debility, anemia, emaciation, with a certain weight and fullness about the stomach, are early but rather indefinite symptoms. Following upon these there occur the classical symptoms: vomiting, pain, perverted gastric function, hemorrhage, development of a tumor, glandular involvement, and cachexia. These are associated with or quickly followed by other phenomena of greater or less significance in the minds of various observers.

The patient may present symptoms so conspicuously gastric in character that our attention is at once directed to the stomach; such cases admit of early and easy diagnosis. Not infrequently, however, the stomach-symptoms are in abeyance, and only the emaciation and debility are in evidence; or, in other instances, we may be misled entirely by the activity of secondary growths in other organs, or the sudden development of acute complications attributable to an unsuspected gastric cancer. It is only by an analysis of the individual symptoms, and ascribing to each its proportionate value that we can arrive at a clear and comprehensive view of the condition, and early master the problem of diagnosis.

Early and Obscure Symptoms: Perversions of taste, anorexia, belching, and pyrosis are minor symptoms that may be regarded as prodromal; in some cases they are entirely absent. In a patient now under my observation the earliest symptoms were a slick, metallic taste in the mouth, constant belching, and vertigo; these were present some six or eight weeks before the characteristic symptoms appeared. The tongue according to many authorities is clean; Ewald refers to the coated condition of the

tongue as being constant. Its appearance is so variable that I do not think it significant.

Pain may not be present until the size of the tumor may become such as to cause pressure, or until ulceration exposes nerves. Ewald says that pain is present in 92 per cent. of cases —absent only in old people. Pain is not peculiar as to location or quality. It is usually somewhat increased by taking food, though it may be several hours before it develops; when it occurs three or four hours after the ingestion of food the pylorus is pointed to as the site of the cancer, and the pain is the result of exaggerated peristalsis. Radiating pains are more common than in ulcer. A pain transmitted under the sternum usually indicates cancer of the cardia. When there is pyloric stenosis and gastric ectasia the patient is always uncomfortable, even in distress, after taking food.

Vomiting: The vomiting is at first of a sour, usually clear liquid. If the cardia is involved, vomiting is usually immediate and small in quantity, for the reason that in cancer of the cardia we have atrophy of the stomach; the food is often returned unmixed with chyme, due to its retention in the dilated esophagus. In pyloric cancer vomiting may be an hour or two after eating; as the case progresses, the vomitus assumes more characteristic features, those of dilatation and stagnation. As Hemmeter says, cancer at the two extremities of the stomach is always attended by vomiting. The patient usually experiences relief from pain, fullness, and distention. Vomiting may not be a daily symptom; it frequently occurs at prolonged intervals. The vomitus then is excessive in quantity, containing the remnants of several feedings, and the quantity seems out of all proportion to the amount ingested.

The vomiting of blood is a fairly constant symptom. In acute medullary cancer there is usually a constant oozing of blood which becomes thoroughly intermixed with the juices of the stomach, and this gives the vomitus the characteristic coffee-ground appearance.

It is impossible to recognize specific cancerous tissue in the vomitus if such be isolated epithelial cells; yet there may be thrown off bits of tumor-tissue which will show the cancerous arrangement (Ewald).

Other special features of the vomitus itself will be considered under their proper headings.

Hemorrhage is usually a rather late symptom, though, as above stated, it may appear early in the acute forms. If ulceration is progressive, large blood-vessels may be opened and we may have copious, even fatal hemorrhage of fresh arterial blood. Of 30 cases noted by McCall Anderson, in 14 hemorrhage was scanty, in 4 free, in 6 very copious. Osler and McCrae found hemorrhage in 36 cases out of 150; in 2 of these cases, vomiting of blood was the first symptom. It may be fairly stated that hemorrhage occurs in about one third of the cases. This refers, of course, to blood evident upon naked eye examination. Occult hemorrhage is present with great regularity constantly in most cases, occasionally in others.

Tumor: Our chief methods of determining the existence of a tumor are inspection, palpation, percussion, and inflation of the stomach. In conducting the examination the patient should be examined in different positions, as change of posture may disclose a hitherto undiscovered tumor; and we must remember the fact that gastric tumors under certain conditions are very movable, and that while the epigastrium is the locality in which they are usually found, they may occupy very different regions of the abdomen.

Inspection: Limiting our field of vision to the stomach-area itself, we may notice unusual fullness about the epigastrium, or active pulsation of the aorta. In about 40 per cent. of cases a distinct tumor is visible. The location of this tumor is in the epigastrium, either in the median line, or to the right if the pylorus is involved. If there is coexisting dilatation, the mass may be much lower than the normal site of the pylorus, and drawn to the left; and here too we observe the asymmetrical contour of the abdomen. Tumors of the anterior wall and greater curvature, though infrequent, are readily seen and palpated.

It is rather broadly asserted that pyloric tumors move only when attached to the liver. In advanced cases with dilatation it is not uncommon for a pyloric tumor to be influenced by respiration, while those of the greater curvature and anterior wall descend with inspiration and ascend with expiration. If the

stomach is distended with food the tumor may be concealed; therefore, many tumors are visible only after lavage. It is equally true that distention of the stomach with air may bring into view a previously unobserved tumor-mass.

Osler has impressed us with the importance of inspection as a method of diagnosis in this disease as well as in many others.

Palpation: In health the triangle between the outer border of the left rectus muscle and the left costal cartilages offers less resistance than the corresponding area on the right side. In early pyloric degeneration, increased resistance in the left triangular region may indicate the development of a tumor. In 80 per cent. of cases a tumor develops at some time in the history.

When the stomach is full, the pylorus may be found pushed two inches to the right of the median line, and overlapped by the lobus quadratus. The lowest part of the stomach is the convexity of the greater curvature to the left of the median line; therefore, only the lower part of the anterior surface is in contact with the anterior abdominal wall. It is evident that the tumor in several parts can be detected by palpation. Cancer of the cardia cannot be felt; so, likewise, cancers of the fundus and lesser curvature escape detection; but cancers of the greater curvature and anterior wall, though rare, when present are easily found. Cancers of the pylorus are readily found notwithstanding overlapping of the liver. Tumors of the curvatures may be pressed upon after inspiration, and their ascent retarded during expiration; this test is of value not alone in distinguishing gastric from other tumors, but in locating the tumor in the curvatures (Ewald). The tumor is often hard and nodular, and two or more separate bosses may be outlined; it is generally immovable. In pyloric tumors, the proximity and frequent adhesions to the liver obscure the boundaries of the tumor, misleading us as to its size.

Sometimes it may be of great advantage to supplement palpation in the dorsal and lateral positions by examining the patient in the knee-elbow posture. Movable tumors will then sink against the anterior abdominal wall and be felt as such.

Percussion: This yields impaired tympany, rarely a flat note. This sign is more misleading than diagnostic.

Auscultation: Aortic bruit may be heard over pyloric tumors, and we also may detect the gurgling or sizzing sound of fluid passing through the stenosed pylorus.

From a diagnostic point of view the presence of a tumor is the most important sign, and Welch's statement of a dozen years ago remains true to-day, that in the absence of a tumor diagnosis is always doubtful. Unfortunately a tumor does not become appreciable until the disease has existed from three to six months, therefore greatly lessening its value as a diagnostic sign in the operative stages of the disease. Other symptoms, in the absence of a tumor, warrant a surgical diagnosis; to wait for the presence of a tumor is to wait too long. Bouveret claims that the presence of a tumor indicates that the disease has passed beyond the limits of the stomach; he therefore upholds Czerny, who maintains that a distinctly palpable tumor is a contraindication to operation. While we cannot accept this fully, yet the prognosis after operation in the presence of a palpable tumor is certainly much less favorable.

Cachexia: The causes of this condition are threefold: (1) *Anemia,* due to insufficient nourishment and hemorrhage; (2) *septic infection* from the tumor itself; (3) *the absorption of leucomains and ptomains* from the retained decomposing stomach-contents. This cachectic state may be briefly expressed as septicemia engrafted upon anemia. All the factors mentioned may not be concerned in an individual case.

As a symptom, cachexia may precede the development of marked gastric symptoms; indeed, as before stated, it may appear very early, and in exceptional instances may be the only evidence of disease during life. It is a conspicuous symptom in the latter half of the case. Rare instances are noted, however, of latent cancer that have pursued their courses without even the manifestation of cachexia, death ensuing from some sudden complication, such as hemorrhage or perforation. The rapid emaciation, the marked debility, the drawn, wrinkled skin of lemon-yellow or grayish-white color, the small crimson patch upon each cheek, standing out sharply against the general sallow hue, hemic heart-murmur, transient edema, exacerbations of temperature, and neurasthenia, are the impressive clinical features of cachexia. Valuable as this symptom is, yet we may exagger-

ate its importance in diagnosis. Other stomachal conditions, innocent in character, produce cachectic phenomena often ascribed to malignancy alone.

Lymphatic glandular enlargement affects the mesenteric, retroperitoneal, supraclavicular, and inguinal glands. Henoch, in 1863, was the first to call attention to the enlargement of the supraclavicular glands in cancer of the stomach. Troisier has emphasized their diagnostic importance. Musser attaches the greatest significance to their enlargement. One might well infer from what he says that upon this sign alone he would feel warranted in making a diagnosis of gastric cancer. Both Osler and Ewald think too much importance has been ascribed to this. We must bear in mind that in tubercular ulcer of the stomach there may be enlargement of the supraclavicular glands; and it is well known that in cancer of the lung, esophagus, and even of the uterus, they may be enlarged. Their presence should merely be accepted as accumulative evidence.

Motor power of the stomach is impaired by the obstruction of the pyloric growth, by cancerous infiltration of the stomach-wall, and by atrophy if the tumor is located at the cardia.

In pyloric obstruction there is for a time a compensatory hypertrophy, during which time active peristaltic waves are observed passing from the left to the pylorus, forming visible patterns. In the height of these spasms the pylorus may come well into view as a contracted hard tumor. Osler, in speaking of the value of inspection as a sign in diagnosticating carcinoma, says that in 36 out of 150 cases peristalsis was visible. In three cases marked movements were visible in the tumor itself. As the obstruction tightens, consecutive dilatation gradually comes on; the vigor of muscular contractions of the walls of the stomach lessens, and in the later months of the disease stomach-peristalsis is not likely to be observed. The retention of food beyond the normal digestive limit is the crucial test of impaired motility.

To determine the expulsive power of the stomach, Ewald's test-breakfast should be employed. After this feeding the stomach should be found empty in two hours. The unreliability of this is that we are never entirely certain that the stomach is empty without repeated washings.

Ewald has also suggested the salol test: 15 grs. of salol are given in three capsules, preferably at the height of digestion. Salicyluric acid is recognized in the urine by the violet color produced on addition of neutral ferric chloride solution. This reaction occurs from sixty to seventy-five minutes after taking the salol. The time of the reaction is subject to such variations as materially to lessen the value of this experiment.

If the propulsive power of the stomach is not impaired, the gastric digestion is largely compensated by the pancreatic juice and saliva, and the patient may not suffer from malnutrition. But the habitual retention of food in the stomach beyond the natural limits gives rise to local pathological changes and general emaciation. It is the loss of this motor power of the stomach that hastens the progress of the disease to a fatal termination.

Boas asserts that when the three factors are constant—absence of free hydrochloric acid, stagnation of stomach-contents, and the formation of large quantities of lactic acid—the diagnosis of gastric carcinoma may be made even in the absence of a palpable tumor.

Riegel concludes that in the gastric juice of a cancerous stomach, as a rule, both free hydrochloric acid and the digestive power are absent; that on the other hand, in any case where free hydrochloric acid and the digestive power are present, carcinoma may be excluded. Voinovitch says that in 19 out of 20 cases no free hydrochloric acid is found in cancer; this statement is based upon 287 analyses. He takes the unwarranted position that the absence of hydrochloric acid is as valuable a sign in cancer as the presence of a tumor in the epigastrium.

Hammerschlag says that in cases of contraction of the stomach, carcinoma may be certainly diagnosed without clinical symptoms by the constant deficiency of hydrochloric acid, excess of lactic acid, with a diminution of pepsin.

Presence of Lactic Acid: Boas concludes: (1) In persons suffering from various diseases of the stomach, especially atony, chronic gastritis, and cicatricial stenosis, non-malignant in character, lactic acid is never found except under very exceptional circumstances, and then it is possible that its origin is in the food. (2) In carcinoma ventriculi we find, with very few excep-

tions, intense production of lactic acid. The positive and repeated demonstration of lactic acid has shown the existence, with almost absolute certainty, of carcinoma of the stomach where no tumor could be found by physical examination. (3) In all cases where excess of lactic acid was found by Uffelmann's reaction, the diagnosis of cancer was verified.

Uffelmann's Test for Lactic Acid: To eliminate any chance of error, it is advisable to give a special test meal as proposed by Boas, to whom belongs the credit of having first called attention to the importance of lactic acid in the early diagnosis of gastric cancer. He advises to give one-half to one liter of moderately thin oat-meal gruel (one tablespoonful to one liter of water), adding salt to the taste, but nothing else; this is to be withdrawn after one hour.

The presence of lactic acid depends upon three factors, the absence of HCl, the presence of motor insufficiency, and lessening of the digestive ferments. This condition is present in 91 per cent. of all cancers of the stomach (Strauss). The same conditions result in the appearance of the Oppler-Boas bacillus.

Uffelmann's is the easiest clinical test for lactic acid. The reagent is prepared as follows: Mix 10 c.c. (two and a half drams) of a three or four per cent. solution of carbolic acid with one or two drops of a neutral ferric chloride solution, and dilute with water until of an amethyst-blue color. This reagent soon decolorizes and it is best to prepare a fresh solution each time a test is made; however, addition of 5 per cent. of chloroform and keeping in a dark bottle may preserve it for some time. Adding some of the gastric contents to this reagent, a canary yellow color will at once appear if lactic acid be present. Lactates give the same reaction as lactic acid.

The presence of sugar, alcohol, citric acid, and certain salts, especially the acid phosphates, give a reaction resembling lactic acid. To eliminate these 5 or 10 c.c. of the filtered gastric juice is treated with ten times its volume of ether free from alcohol, shaken for fifteen or twenty minutes in a stoppered separating funnel; after the layers have separated, the ethereal solution is allowed to evaporate, the residue is dissolved in 5 to 10 c.c. of water, and this solution tested for lactic acid as above.

The Boas qualitative test for lactic acid, while more delicate

than the above, is too complicated for clinical purposes, and the quantities of lactic acid which have any pathological significance can readily be detected with Uffelmann's test.

Absence of Hydrochloric Acid: The diagnostic value of the absence of hydrochloric acid is universally admitted. Hemmeter claims that only in carcinoma arising from ulcer do we find it. Upon the other hand, Rosenbach opposed the views of Boas and Riegel some time ago, and says that his later experience has but strengthened the opinion he then expressed. He does not consider the absence of hydrochloric acid as of any value in the diagnosis of gastric cancer. This is an extreme position and is not sustained. Robin, "although regarding the absence of hydrochloric acid as a diagnostic sign of great value," reports 12 cases in which cancer of the stomach was accompanied by excess. He further argues that the size and situation of the growth affords an explanation for this; if it remains limited to the pyloric region, the greater part of the gastric mucous membrane is uninvolved and performs its function normally; if before the appearance of cancer there was excess of acid, in other words, if there is preceding ulceration, the presence of hydrochloric acid will persist to the end. In recent literature numerous cases are reported in which free hydrochloric acid is present; hence we must conclude that Ewald is correct when he says that absence of hydrochloric acid in cancer of the stomach has no especial significance as such, but only has value as a secondary symptom.

It has been demonstrated by Moore, of Liverpool, that the absence of free hydrochlorate acid is not characteristic of gastric carcinoma alone. According to Moore, it is absent in two thirds of all carcinomata wherever situated, and is markedly reduced in the remaining one third.

The tests for hydrochloric acid have been given in the chapter on Ulcer of the Stomach.

Pepsin and rennin are not lessened in the same proportion as hydrochloric acid. No practical diagnostic results have thus far been gained by tests for the gastric ferments in gastric cancer.

On microscopical examination the vomitus is rich in bacteria, and contains the Oppler-Boas bacillus; this is characteristic of lactic acid fermentation, but is not pathognomonic of cancer, and too much confidence ought not be placed in this.

The diagnostic significance of lactic acid is thus well expressed by Ewald: "The presence of lactic acid is as little specifically characteristic as the absence of free hydrochloric acid. Lactic acid only occurs in gastric cancer after serious alterations of digestion have appeared, and usually after a palpable tumor is present."

Solomon's test for albuminous secretion from a tumor of the stomach gives positive results with simple ulcer, while Maragliano's biologic reaction is not specific. Even if these tests were reliable, they would only be available in the late stages of the disease.

Blood Changes: R. Lépine is the first to point out a diminution in the globular richness of the blood; it is, of course, a late sign. Hemoglobin may be as low as 35 per cent. to 40 per cent. in cancer, while in pernicious anæmia it is conserved or even augmented. The average blood-count is about 3,000,000 to 4,000,000; if it falls to 1,000,000 or lower, it points to pernicious anemia and not to cancer.

There is increase of white cells usually; this may be due either to hemorrhage or to ulceration.

Referring to digestive leucocytosis, Carstairs Douglas says that he is able to report 11 cases of undoubted malignant disease of the stomach, in which diagnosis was confirmed by one of several ways. He regards the leucocytosis-test a faulty method, and regrets that his criticism is destructive in character to this supposed sign.

Bowels: The bowels, as a rule, are constipated; in the advanced stages of the disease, especially when attended with ulceration or sloughing, a septic diarrhea may develop. This presages an early termination.

Edema: Boas first called attention to the local edema about the ankles as an early and transient symptom; this lacks the confirmation of sufficient observation. In the later stages of the disease, ascites or anasarca frequently appears. This cachectic dropsy is due to hydremia.

Phlebitis: Varicosities of the veins in the inguinal and crural regions are frequently present, and Trousseau attached the greatest importance to the appearance of phlegmasia alba dolens. These signs have no especial value, as they occur in many other intra-abdominal conditions.

Metastases: Babes and Stoicesco have discovered that the microscopical examination of small subcutaneous, metastatic tumors may be of considerable value in diagnosing cancer of the internal organs. Osler refers to the detection of these by inspection, especially around the umbilicus, as significant of cancer of the stomach.

Metastatic growths appearing in other organs, in the liver especially, may mask the symptomatology of the primary disease. Metastasis occurs earlier in the more acute forms of cancer, and in young individuals. The extreme emaciation permits of easy detection of enlarged lymph-nodes and secondary growths. It has been remarked that nodules may be felt in Douglass's cul-de-sac; this is not peculiar to cancer of the stomach.

Coma Carcinomatosum: This is a complication or late symptom, accompanied by a peculiar dyspnea, respiration strong and deep, and accompanied by groaning, temperature often subnormal. It is a sign of toxemia (Hemmeter).

SUMMARY OF SYMPTOMS: In reviewing the symptomatology of carcinoma, the salient features present themselves in the following order: The patient is usually a male between the ages of 40 and 60 years, without history of digestive disorder, and suddenly develops perversions of taste, has eructations of gas, pyrosis and anorexia; is losing flesh, complains of weakness and general debility; has a sense of uneasiness and fullness in his stomach which amounts to pain, usually increased 2 to 4 hours after eating; vomiting now appears, first due to irritation, later to ischochymia and dilatation. Hemorrhage, moderate or copious, may supervene. There is now, usually, pronounced cachexia. From three to six months after the initial symptoms a tumor may appear, as a rule, at the site of the pylorus. Motor disturbances and dilatation of the stomach now become marked features. Examination of the stomach-contents usually shows absence of hydrochloric acid and presence of lactic acid. The lymphatic glands are enlarged, metastatic growths appear, constipation may now be followed by a diarrhea, there is slight septic fever and dropsy. The terminal stage of the disease is one of marasmus, the patient really dying of hunger and thirst; coma or delirium marks the closing hours.

DIFFERENTIAL DIAGNOSIS.—**Ulcer of the Stomach:** While age and sex guide us somewhat in distinguishing ulcer from carcinoma of the stomach, the occasional occurrence of carcinoma in the young and in females nullifies to a degree this differentiation. A pylorospasm, though transient, may be mistaken for the tumor of carcinoma. If paragastritis occurs the massing adhesions may simulate a malignant tumor. The vomiting in both ulcer and cancer may not immediately follow the ingestion of food. In gastric cancer the vomiting is that of pyloric obstruction and retention; not so in ulcer except in the late stages with complications. In pyloric ulcer the pain and vomiting may not occur for two or three hours; but it is relieved more decidedly when the stomach is empty in ulcer than it is in cancer. In cancer the blood in the vomitus is of a characteristic coffee-ground appearance; the hematemesis in ulcer is more profuse and of fresher red blood. Loss of appetite is characteristic of cancer; hunger, of ulcer. The course of carcinoma is more rapid, the general health is more impaired, cachexia, metastases, and glandular involvement appear. Examination of the stomach-contents, showing the presence of lactic acid and the absence of hydrochloric, is practically conclusive for cancer. Exceptions to this rule are quite rare.

The absolute unreliability of all clinical and laboratory evidences of gastric carcinoma early in its course, when there is hope of permanent relief from surgical treatment, makes exploratory incision more or less essential in patients of the cancer age (after 40 especially), whenever digestive trouble associated with stagnation of stomach contents refuses to respond to careful medical treatment. When we have in addition a history of previous ulceration and marked impairment of the patient's general health, the indications for exploration are imperative. Musser lays stress on the importance of a family history of cancer in these cases.

PROGNOSIS.—**Course:** The course of cancer depends: 1st, upon the age of the individual; 2d, upon the histological character of the growth; 3d, upon the site of the tumor. The younger the individual, as a rule, the more rapid the course of the disease. Neoplasms rich in cellular elements, the medullary and cylindrical-celled varieties, are more rapid in growth, more liable to destructive changes, and show greater tendency to hemor-

rhage, perforation, and metastasis. Cancers of the cardia and pylorus, by their obstructive character, interfere with nutrition and quickly lessen the vital resistance.

Duration: The duration of cancer is so variable that it is scarcely possible to place an estimate, but the majority of cases terminate within a year.

TREATMENT.—The treatment of cancer of the stomach is essentially surgical. With an operative mortality already reduced in skilled hands to 10 per cent. and with 25 per cent. of operative recoveries living more than three years, partial gastrectomy offers an increasingly brighter outlook to these sufferers. In 80 per cent. of cases of cancer of the stomach the disease is situated at or near the pylorus, and if discovered early, is amenable to removal by this operation. Improvement in permanent results of operative treatment depends upon early diagnosis, and this in the present state of our knowledge on exploration in suspected cases. To await positive clinical and laboratory proof of the presence of cancer is to allow the patient to pass beyond any material hope of permanent cure.

Metastases, the involvement of adjacent structures, the presence of ascites, or a profound cachexia, are contraindications to gastrectomy.

Palliative operation, gastrostomy, gastroenterostomy and jejunostomy may be indicated at times when the more radical procedure is impossible of accomplishment. These palliative operations, being done in the worst cases, are attended by a higher mortality than gastrectomy. The prolongation of life in this way does not average over five months, but the relief of urgent symptoms and the prevention of starvation will at times necessitate their performance.

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SARCOMA OF THE STOMACH.

The great rarity of sarcoma of the stomach, and the similarity of its symptoms to those of carcinoma, render an exhaustive study of it unnecessary.

Howard's statistics show that there are 61 cases reported in the literature up to 1902. Staehlin (1908) collects 82 cases from literature and reports one of his own.

Sarcoma of the stomach may be either primary or secondary; like carcinoma, it is usually primary. As a rule, it affects the greater curvature, appearing in three forms: as myo-, fibro-, and lympho-sarcoma. The lympho-sarcoma originates in the muscular or submucous structure of the stomach. The mucous membrane becomes secondarily involved. In many cases the tumor extends into the peritoneal cavity; this, however, is especially true of the myo-sarcoma. It attains to considerable dimensions, usually much larger than carcinoma. In Baldy's case it filled the abdomen from ensiform to pubis; in Cantwell's, the tumor removed weighed twelve pounds.

These tumors are usually round, knotty, and irregular, and situated on a small base. Metastases in the neighboring organs and especially in the lymphatic glandular structures appear. In four cases of lympho-sarcoma observed by Kundrat, the neoplasm infiltrated the entire stomach; but it is usually confined to the pyloric portion.

Harlow Brooks reports a case as developing after a gunshot wound; this case is unique in its etiology. It occurs in all ages, but most frequently in those of from twenty to thirty-five years. Men are perhaps more frequently affected than women.

Symptoms are often obscure. This is accounted for by the fact that the growth tends at times to project toward the peritoneal surface and may even be pedunculated. Hemorrhage rarely occurs until late in the disease. Peritonitis is more common. In those forms that are seated at the pylorus the obstructive symptoms and chemical analysis of the stomach-contents are practically the same as in carcinoma. Schlesinger found swelling of the spleen in most

cases, and he regards this as an important diagnostic sign, differentiating it from carcinoma. The tonsils and follicles on the side of the tongue enlarge. Cachexia is rapid and progressive. Slight pyrexia is an early symptom, high fever usually due to complications.

The duration of the disease may be only a few months without surgical treatment, but it may last two or three years; the average is about nine months. It shows, as does sarcoma elsewhere, a pronounced tendency to metastasis. The treatment is distinctly surgical, and gives a better prognosis than carcinoma, especially with regard to local recurrence. The operation indicated is partial gastrectomy or resection of the stomach, palliative operations, gastroenterostomy, jejunostomy, etc., are at times necessitated.

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PYLORIC OBSTRUCTION.

DEFINITION.—A partial occlusion of the lumen of the gastro-duodenal opening, retarding the discharge of chyme, and attended by certain definite pathological changes in the stomach itself, to which complications its symptoms are largely due.

FREQUENCY.—When we analyze the number and variety of the conditions that may produce pyloric obstruction, we can appreciate the frequency of its occurrence.

ETIOLOGY.—A general classification of the causes of pyloric obstruction may be made under two heads: (1) *intrinsic*, (2) *extrinsic*. The former are by far the more frequent, and comprise such conditions as neoplasms, benign and malignant, congenital hypertrophic stenosis, fibroid degeneration of the pylorus, cicatrization of pyloric and duodenal ulcers, spastic contraction of the pylorus due to hyperchlorhydria, pouches or diverticula of the mucous membrane. The *extrinsic* causes are: adhesions secondary to pyloric and neighboring inflammations, constriction at the gastro-duodenal junction due to gastroptosis, especially effective when there is a short gastro-hepatic omentum, pressure from gall-stones, pressure exerted by tumors of some of the neighboring structures not connected with the duodenum or stomach. Page and Clay report a case produced by kinking, the result of adhesions of a suppurating gall-bladder, shown by post-mortem examination. Glénard pointed out that the duodeno-jejunal junction may be compressed by traction upon the mesentery. Kundrat describes several cases in which he found the stomach and duodenum greatly dilated, the small intestine being crowded down into the pelvis; this cause is especially active after celiotomy by reason of the temporary loss of intra-abdominal pressure, and is of immense surgical importance.

As we have seen, carcinoma of the stomach occupies the pyloric region in the majority of cases. The cylindrical-celled and medullary carcinomata may form distinct tumors that project into the lumen of the pylorus, mechanically occluding its opening without materially contracting it. The scirrhous form almost

invariably involves the pylorus, and since its nature is to infiltrate and contract the involved part, it produces pyloric stenosis more frequently than all other forms of carcinoma.

Hyperchlorhydria itself, with or without ulceration, may produce spastic contraction of the pylorus. Of nearly 100 operations for pyloric obstruction, in 46 Doyen found stenosis of the pylorus due to spastic contraction without cicatrization or other changes. While we do not discredit the work of this eminent surgeon, yet functional disorders rarely require surgical intervention.

Ulcers of the pylorus may run their course, and contraction of their cicatrices, producing pyloric stenosis, may be the only evidence of their existence. Ward's patient lost 72 pounds in seven months from pyloric obstruction due to the healing of an old ulcer.

Dr. Pertik describes a remarkable case of a finger-like reduplication of the duodenal mucous membrane occluding the pylorus and producing dilatation of the stomach. Deiters has collected quite a series of these congenital malformations producing obstruction and consequent dilatation.

Instances are reported of adhesions of the gall-bladder, with stones incarcerated in the cystic duct, producing pyloric obstruction, of which A. A. Smith's case is an excellent example. It is unnecessary to further detail the numerous causes.

PATHOLOGY.—The pathology of pyloric obstruction is peculiar to the various causes, but loss of motor power and consecutive dilatation are the inevitable sequences.

SYMPTOMS.—As we have seen, pyloric obstruction is the outcome of such a variety of causes, necessarily the symptoms must vary with the pathogenesis. It is of greatest importance to investigate exhaustively the history of every case presenting the symptoms of pyloric stenosis. The treatment is essentially surgical, and that surgeon does the best work who best connects his symptomatology with definite pathology. It is not sufficient to recognize merely a stenosis; this calls loudly for operative relief, yet there may be associate conditions which forbid even the consideration of the expediency of it.

The gastric distress, the vomiting of irritation, in the majority of cases the presence of a tumor, with motor disturbances and

the development of increasing dilatation with its characteristic symptoms, the rapid emaciation, the obstinate constipation, diminished urinary secretion, and distressing hunger and thirst are all symptoms of the greatest value, but there is but one essential symptom of pyloric stenosis, and that is the presence of residual food in the stomach beyond the normal limits of digestion—this must be present before an absolute diagnosis of stenosis can be made.

PROGNOSIS.—We may forecast the course and duration of the disease with some accuracy if we can determine the benign or malignant character of the obstruction; yet every case of persistent stenosis pursues a definite downward course leading to a fatal termination. Spastic contractions are essentially intermittent, and are not necessarily fatal in their termination.

The surgical indications in pyloric obstruction depend upon the pathology of the given case. So far as relief of symptoms due to obstructions are concerned there is no more satisfactory field of work in abdominal surgery, and this feature is fully met by the performance of gastrojejunostomy. Unfortunately in many instances, especially in malignant obstruction, this operation is merely palliative and more radical measures are necessary for the permanent cure of the condition present.

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CONGENITAL HYPERTROPHIC STENOSIS OF THE PYLORUS.

DEFINITION.—A congenital spasm of the pyloric area, resulting in hypertrophy and stenosis of the pylorus.

FREQUENCY.—Thomson says that there have been only 45 reported cases, one-half of these within the last three years (1897). Up to May, 1905, Scudder and Quinby collected 115 cases, 55 autopsies and 60 operations. Thirty odd cases are on record in which the symptoms were sufficient to lead to a correct diagnosis. Maier has collected 31 cases in the autopsy-room which he considered congenital; he bases his opinion upon the absence of pathology that could cause hypertrophy and stenosis. As the youngest was 12, and the oldest 80, it is not sure that these cases were all congenital, yet it is admitted that some cases of congenital stenosis not only survive infancy but reach advanced life. Evidence is accumulating that this abnormality is far more common than was hitherto supposed.

ETIOLOGY.—Nicoll gives the following theories of the causation: (1) that the condition is the result of a congenital developmental aberration, possibly connected with the junction at the pylorus of two different processes of development; (2) that congenital hypertrophy is secondary to antagonistic incoördinated action of the muscles of the stomach on the one hand, and those of the pylorus on the other, dependent upon functional disorder of the gastric nervous system, ultimately resulting in congenital gastric spasms; (3) that it is due to gastric dyspepsia arising after birth, and hence not congenital at all; (4) that it is due to a chronic inflammation.

Thomson considers the primary cause to be a nervous disturbance leading to spasm or incoördination of the muscles of the pylorus, the hypertrophy of the muscle being the natural consequence. Pritchard, also, regards the spasm as primary, and the hypertrophy and the stenosis secondary.

It must be remarked that Maier adduces that the closure of the foramen Winslowii might be an etiological factor (?).

Heredity: The subject is entirely too new to determine the question of heredity, but Henschel records three cases occurring in one family.

Sex: There is no manifest predilection for either sex. It was perhaps a mere coincidence that 17 of Still's 20 cases were males.

PATHOLOGY.—This condition was first described by Landerer. Microscopically the hypertrophy may involve both muscular coats, or the circular only, but usually there is a combination of muscular hypertrophy with fibrous thickening. In three cases Gran found only the circular fibers involved; they were hypertrophied and gathered into larger bundles. Schwyzer found the mucosa greatly, the mucularis enormously, and serosa somewhat thickened, the chief part of the tumor being formed by the circular muscular fibers. Still describes a case of great thickening of the muscular wall, but the lumen was normally patent. Meltzer reports a case of congenital stenosis with no pathology at all. In no case has there been atresia; in some the pyloric end of the stomach invaginates into the duodenum. The stenosis is chiefly due to spasm, and, as measured at post-mortem, is but an inaccurate gauge of stenosis in life; in proof of this, Pfoundler states that in congenital stenosis at autopsy the pylorus is found contracted, but after repeated washings with water, or even after waiting a few hours, it will be found of normal size.

Kammerer says there are two forms of congenital stenosis: (1) absolute at birth, (2) that which is progressive, but not primarily absolute.

The constant pylorospasm with consequent compensatory hypertrophy of the muscular wall leads on to degeneration. Over-exertion, assisted perhaps by malnutrition, favors the development of the dilatation, so that spasm, hypertrophy, and gastrectasia form the pathological cycle.

SYMPTOMS.—The average age when pyloric stenosis is recognized by symptoms, according to Rolleston, is about three months. A study of recorded cases shows that it is rarely earlier than this. In 52 cases the average period after birth when vomiting, the first symptom, occurred was seventeen days. In Wachenheim's table of 35 cases, in 7 the vomiting started at birth; in others it was from three days to six weeks. The term *congenital* is therefore somewhat misleading. The onset of symptoms depends upon whether

obstruction is absolute or partial. Vomiting first appears only once or twice a day and may not attract particular attention from the parents or nurse. It increases in frequency and has the following characteristics: Not influenced specially by the character of food, but usually occurs soon after feeding, whether from the bottle or breast; it is copious and is expelled forcibly (pumped out), often appearing through the nostrils. The child may go twelve to twenty-four hours without vomiting, then the amount ejected will be the accumulation of several feedings; the vomitus is acid and rarely contains bile; constipation is quite the rule, though diarrhœa has been observed. When the obstruction is absolute the stools are like meconium. The child is ravenously hungry; takes food eagerly, but is not comfortable after feeding and is restless until it is rejected; there is rapid loss in weight. A child presenting these symptoms should be subjected to careful physical examination. The appearance is in itself almost diagnostic. The wasted thorax, the deep intercostal spaces, the protuberant epigastrium, the flat sunken hypogastrium, is a characteristic picture. In advanced cases the emaciated condition of the patient renders visible the outlines of the dilated stomach and gastric peristaltic waves, originating under the left costal margin, passing from left to right to disappear in the right hypochondrium. This exaggerated peristalsis is to be best observed during or soon after taking food. In a typical case a pyloric tumor may be felt; in many instances the liver overlaps the pylorus and prevents its palpation. The tumor, like that of intussusception, is only palpable when the muscle is in spasm. To the touch it is an insensitive, hard, movable, barrel-shaped lump about three fourths of an inch wide. The resistance offered to the touch varies from time to time as the muscles contract and relax.

Summary of Symptoms.—A history of vomiting in the early weeks of life, progressive emaciation, visible gastric peristalsis are sufficient for diagnosis and the evidence is conclusive when supported by a palpable pyloric tumor.

Prognosis.—In the light of the statements of Maier, and Landerer we must assume that some cases of congenital stenosis not only survive infancy but reach advanced age; yet when the disease reaches the stages of hypertrophy and dilatation, a fatal result may be expected.

TREATMENT.—Congenital hypertrophic stenosis of the pylorus is not necessarily a surgical condition. Moynihan states that "incredible as it may seem, cure will sometimes be achieved by medical measures." It is impossible, of course, to absolutely establish the diagnosis in cases recovering without operation, but the clinical evidences are certainly clear enough in most cases not to require exploratory confirmation. The important features of the medical treatment are rest for the stomach (lavage is not used), warm applications to the belly wall and administration of atropine and opium by rectum. If improvement is not prompt surgical treatment should be substituted. Posterior, no loop, suture gastrojejunostomy is probably the operation of choice. Scudder and Quinby quote the following operative results: From 40 gastroenterostomies there were 21 recoveries and 19 deaths. From 11 Loreta operations there were 7 recoveries and 4 deaths. From 8 pyloroplasties there were 4 recoveries and 4 deaths. One pylorectomy, 1 death.

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DILATATION OF THE STOMACH.

DEFINITION.—An abnormally large stomach with deficient motor power.

SYNONYMS.—Gastrectasia; ischochymia (Einhorn).

FREQUENCY.—Dilatation occurs as a sequel of such a variety of causes that every general practitioner can attest to its frequency.

ETIOLOGY.—The causes of dilatation are numerous; they may be comprehensively grouped under two heads, (1) *atonic,* (2) *obstructive.*

1. **Atonic:** As the term atony implies, the essential feature in this condition is a weakening of the muscular walls. It is the outcome of local and constitutional causes; the local causes are chiefly attributable to errors in diet, over-eating and drinking, and chronic gastritis; the constitutional conditions are the various neuroses, malnutrition, and general debility. Atonic dilatation does not exist as a pathologic entity, but is often a conspicuous local phenomenon of a general disease.

Acute dilatation of the stomach is usually atonic in nature. Walter D. Laffer, of Cleveland, Ohio, has collected and analyzed 217 reported cases (1908). It is most frequently met with as a sudden, unanticipated, and rather late complication of severe injuries or extensive surgical operations, not necessarily connected with the abdomen. It is also attributable to gross errors in diet, and as a complication of constitutional conditions marked by great debility.

Duodenal compression and constriction is emphasized as a frequent cause of dilatation occurring after abdominal section. Glénard and Albrecht assert that as after celiotomy the intra-abdominal pressure is lost, there is general enteroptosis, and the traction exerted upon the superior mesenteric artery with its accompanying bundles of connective tissue, compresses the duodenum and causes stagnation of stomach contents. The author has taken some pains to verify this mechanical theory, and is disinclined to accept it as the essential factor in the gastric distention.

2. **Obstructive** or **Consecutive**: This is a sequel of the various conditions producing pyloric stenosis: malignant diseases, irritable ulcer, cicatricial contraction, pylorospasm due to hyperchlorhydria, peripyloritis, extraneous pressure from tumors, gallstones, etc.

Age: Atonic dilatation occurs most frequently in infancy and in advanced life. Acute dilatation is most frequent between the ages of 20 and 30. Comby asserts that dilatation of the stomach in children is by no means rare, and that its association with rickets is not a mere chance occurrence; they both are dependent upon improper alimentation. The general debility of old age, associated with local causes, may give rise to gastric myasthenia. The obstructive types are more common in middle and advanced life.

PATHOLOGY.—In atonic dilatation arising from constitutional causes, the muscular walls are found of normal thickness or of unusual thinness (Ewald). As the result of food-stagnation and fermentation we find gastric catarrh. We therefore have gastritis both as cause and effect of dilatation. Occasionally, in the dilatation found in young and poorly-nourished children, pectiniform ulcerations and capillary hemorrhages are observed in the mucous membrane at autopsy. In acute paralytic conditions the stomach looks like a "tightly-distended cylinder, shaped like a V" (Thomson). It is distended with gas and fluid, without macroscopical changes in the wall.

In obstructive dilatation there is first compensatory hypertrophy of the muscular wall, then dilatation with thinning. The dilatation is due to the existing inflammation and degeneration of the muscular fibers, yielding under the long strain of an overdistention from the stomach-contents. At the pylorus is found the prime or causal pathological condition.

SYMPTOMS.—In explanation of the symptoms we must bear in mind the impressive statement of Welch, that there are three inevitable results of dilatation: loss of motor power, disordered digestion, and arrested absorption. Investigation along these lines will develop the symptomatology.

As dilatation of the stomach is the product of some condition local or remote, it is imperative that we should obtain, by searching interrogation, the history; this will not only determine the

obstructive or atonic character of the dilatation, but will guide us in our prognosis and therapy. It is obvious that the symptoms due to cancer of the pylorus would be very different from those of atonic dilatation following a protracted illness, or consequent upon severe injury or operation. Every case will present not alone the symptoms of dilatation, but the symptoms of the prime cause. It is not sufficient, therefore, to determine simply the presence of dilatation, but the cause also must be determined, and oftentimes this can only be done by a process of exclusion.

In order to recognize dilatation, every method of diagnosis must be employed. The patient usually complains of dyspepsia, indigestion, of weight or fullness, employing some such indefinite terms to direct our attention to the stomach.

Pain: The existence of actual pain depends upon the cause of the dilatation. In simple atonic dilatation the pain is never acute, although the distention may be distressing. In obstructive dilatation we may have the pain peculiar to ulcer or cancer, aggravated by tension upon the diseased pylorus. Pain and distress are usually relieved by vomiting.

Vomiting: This is the most characteristic symptom of dilatation, and it possesses diagnostic peculiarity. It occurs usually late after taking food, indeed, often bears no relation to the time of taking food. If there is marked associated gastritis, early in the history there may be frequent vomiting, but as the case reaches an advanced stage of relaxation, the vomiting becomes infrequent, at irregular intervals, the distinguishing feature being the quantity and character of the vomitus; there is an enormous amount ejected (as much as 17⅗ lbs. is recorded), and it contains remnants of food taken hours or even days before. This retention of food in the stomach beyond the normal limits for stomach-digestion, which Leube has fixed at seven hours, is in itself conclusive proof of motor deficiency, an essential element of dilatation. The vomitus has a brownish-green color, offensive odor, and a floating scum. As Hochhaus has emphasized, if the stenosis is in the duodenum, below the ampulla, there is usually a large quantity of bile in the vomitus; this is especially true in post-operative dilatation. In all forms of dilatation after copious vomiting the abdomen is flat, and there is usually marked relief experienced.

HCl is usually absent in malignant stenosis and in dilatation from chronic gastritis; in old cicatrized ulcer, the form that usually gives rise to dilatation, it may also be absent from exhaustion of the gastric glands.

Ewald says that entirely too much importance is attached to the presence of sarcinæ.

The uncertainty or unreliability of the potassium-iodid test for the absorptive power of the stomach, and of the salol- and oil-tests for the motor power, renders these of small value in the diagnosis. These tests will be found under Carcinoma of the Stomach.

Notwithstanding the discomfort and vomiting, the patient retains a good appetite. He may be ravenously hungry at times, and complains of great thirst. His tongue is usually clean, though the heavy coating characteristic of gastritis may be present. His breath is offensive, he eructates foul-smelling gas constantly, in a noisy, prolonged effort; this belching is due to the fermentation of the stagnating stomach-contents. Constipation is obstinate, the stools are dry, scybalous masses. The urine is scanty, malaise is marked, and headache is a constant accompaniment. Vertigo is a frequent symptom. If the distention is great the patient complains of cardiac embarrassment, has palpitation, feeble, irregular pulse, and often precordial pain, which may be mistaken for angina. The peripheral circulation becomes decreased, shown by moist and cold extremities. Broadbent lays much stress upon the nocturnal asthma; this is due not only to pressure, but to the toxemia. There may be also some slight toxic temperature.

From the retention of food and the non-absorption by the gastric and intestinal mucous membranes, there is produced an abnormal dryness of all the tissues of the body, as expressed in deficient urine, constipation, and dry skin. Kuckein believes that the thickening of the blood resulting from this non-absorption produces a symptom very common in gastrectasia, namely, tetany; this is a nervous phenomenon which manifests itself by painful spasms of the flexor muscles of the arms and legs, and of the abdominal muscles; these attacks usually begin in the region of the stomach, and may last some hours. In them the face assumes a rigid mask-like appearance. The temperature rises

and the pulse becomes weak and feeble, and not infrequently the patient dies in the attack. If he survives the attack, it may recur at irregular intervals, usually after severe vomiting. Kussmaul was the author of the idea that these phenomena proceeded from the dryness of the tissues, but now believes that the chief cause of gastric tetany is auto-intoxication.

The infection from decomposition produces nervous depression, the patient's sensorium is clouded, he is melancholic and drowsy, and may pass into a stupor, very aptly called *coma dyspepticum,* in which state he may make his exitus.

During the history of gastrectasia, or in other forms of stomach disease, there not infrequently develops a tetanoid state; but true gastric tetany is rarely seen except with dilatation of the stomach. At first it may appear as simple cramp in some of the muscles of the limbs, soon after returning; and in one patient, with whose history I am quite familiar, there appears first a feeling of numbness of the extremities, throbbing in the temples and a sensation of swelling in the head. These are preceded for some hours by acidity and belching. In the milder cases the phenomena usually appear at night. The patient is conscious of gastric discomfort, has pyrosis and belching, then is restless and nervous; complains of tingling sensation in the hands, has vertigo, throbbing temples and his head feels swollen. These symptoms are usually relieved by vomiting, voluntary or reduced. Cramping in the muscles, especially of the extremities, is now noticed and there is an inclination to extend the limb as far as possible. In the milder cases the cause is entirely overlooked and the phenomena ignored. In the more severe cases the seizure may overtake the patient at any time. The contraction of the muscles of the hand throws it in the position of the accoucheur's hand; all the muscles of the body may be involved in the tetany and, as Trousseau has written, "so long as the attack is not over, the paroxysm may be produced at will. This is effected by simple compression of the affected parts, either in the direction of their principal nerve trunks or over their blood vessels, so as to impede the venous or arterial circulation." Chvostek's sign is the increase in the mechanical excitability of the motor nerves. Hofman's symptom is exaggerated excitability of the sensory nerves. There are evident, varying degrees of gastric tetany; but as a symptom we cannot overestimate its gravity. It has been the immediate

cause of death in two patients under my observation suffering from malignant pyloric obstruction. In acute dilatation we have vomiting, gastric discomfort, depression as shown by rapid pulse and subnormal temperature, abdominal distension with stomach splashing, constipation, scanty urine and severe thirst.

PHYSICAL SIGNS.—In entering upon an effort to make a diagnosis of dilated stomach, we must bear in mind that the normal position of the stomach varies greatly. Ewald has emphasized the frequently vertical position. Luschka places the pylorus in the triangle between the right border of the xiphoid cartilage and the right costal margins. The lower border of the greater curvature is well above the umbilicus. The fundus lies beneath the base of the left lung and is almost covered by its projecting margins. The relations of the stomach are somewhat changed with the posture of the patient.

While we attach the greatest importance to the subjective symptoms of gastrectasia, physical signs are essential to the establishment of a diagnosis. If Osler's classical work upon Abdominal Tumors were accessible to all students, further contribution upon this subject would be unnecessary.

The General Appearance of the Patient: The marked emaciation, the anxious, hungry expression, sunken eyes, dry, yellow parchment-skin, and cracked mucous membranes, simulate cachexia.

Inspection is one of the most valuable means of diagnosis. The asymmetrical distention of the abdomen is suggestive; the right hypochondrium is usually flat; the epigastric, but especially the umbilical and left hypochondriac regions are usually markedly distended. In moderate dilatation the enlargement of the abdomen may not be noticed as long as the patient retains the recumbent posture, but becomes apparent when the patient stands.

The statement of Musser and Steele, that inspection is negative, is surprising. It is true in atonic dilatation; the flaccid walls of the stomach accommodate themselves to the abdominal cavity and the outlines do not stand out distinctly, nor is there visible peristalsis; but in dilatation of obstructive origin not only can we oftentimes outline the borders of the stomach by inspection, but waves of peristalsis from left to right, terminating in

the epigastrium and often developing a tumor at the pylorus, appear. The lesser curvature of the stomach, says Osler, is often much more distinct in outline than the greater, its line passing vertically. The greater curvature descending on the left side, according to the degree of dilatation, occupies some position between the umbilicus and pubis, then curving to reach the pylorus, may extend well into the right side. These borders are distinct when peristalsis is excited. I have occasionally seen the pyloric region in obstructive dilatation rise up as a large globular tumor, and then slowly relax.

The value of inspection must be appreciated, since Osler made the diagnosis by this sign alone in 10 out of 13 cases, but its special value is in obstructive dilatation.

Palpation: A pyloric tumor may be felt, usually displaced downward and often very movable. Manipulation may excite peristalsis, the stomach then offering a changing resistance. Palpation combined with percussion will elicit "splashing"; while this is a valuable sign we cannot attribute to it the diagnostic value that Armstrong assumes for it.

Auscultatory Percussion: This method of diagnosis is really the most valuable of all in outlining the size and position of the stomach.

The stomach should first be emptied of its contents, and then be inflated with atmospheric air through a tube, or else distended with gas, the patient taking a teaspoonful of tartaric acid in water, followed by a somewhat larger quantity of sodium bicarbonate. After inflation the outlines may become visible; the pyloric tumor may disappear, or become more prominent according to its location. We must bear in mind that the area of tympany varies with the amount of stomach-contents, hence the advisability of lavage before inflation.

Auscultatory percussion is more accurate and delicate than simple percussion. With the stethoscope placed over the center of the stomach we very lightly percuss with a slight glancing blow, beginning close to the stethoscope; we thus ascertain the normal stomach note, and percussing progressively outward in each direction, we are at once aware of the change of note when the borders of the stomach are passed.

In extreme dilatation, owing to the increased weight and relax-

ation, the lesser curvature may lie surprisingly low, reaching to midway between the umbilicus and ensiform cartilage, and, as before stated, may occupy a rather vertical position. After inflation auscultatory percussion should be employed in both recumbent and standing positions. In percussing a stomach without previous lavage, we must remember that the greater portion is tympanitic, and that the lower part is dull when the patient is standing; when we change the position of the patient the resonance will vary. If the stomach dulness in the standing position is below the umbilicus, there is either marked gastroptosis, or else dilatation, probably both. Concerning the change in the area of dulness, König says that in a dilated stomach, when the patient lies on the back, there is dulness in the left lateral region, which dulness disappears when the patient is turned on the right side. Ferber has called attention to the fact that the circular tympany, "stomach-lung region," formed by the stomach under the lower border of the left lung, gradually disappears behind the axillary line if the organ be normal, while in dilatation it may be traced back to the vertebral column. These signs, of course, depend upon the amount of fluid or ingesta in the stomach.

Here I would again emphasize that the physical examination of the abdomen should be preceded, when practicable, by lavage; by this means we not only detect the presence and estimate the amount of residual food in the stomach, but we also prepare the organ for inflation. If inflation is not available at the time, we have a useful aid in determining the capacity and outlines of the stomach in Dehio's method; to employ this method properly we must here, too, thoroughly wash the stomach, then require the patient, while in a standing position, to drink measured quantities of water; then by simple percussion, or preferably by auscultatory percussion, from the lower limits of dulness upward we outline the greater curvature of the stomach. We may also test, to some extent, the capacity of the stomach, knowing the normal, which Ewald places at 1500 c.c., and Stengel at 2600 c.c. The amount of fluid that a patient will take is by no means a guide to the capacity of the stomach, however.

Aufrecht lays stress upon two signs: (1) Variation in percussion note—a gentle blow over the stomach in the same situation will at one time be dull, at another tympanitic, by reason

of the more voluminous and energetic peristaltic contraction of the hypertrophied wall of the stomach; (2) cracked-pot sound, best elicited at the junction of the area of circumscribed dulness and the adjacent tympanitic area—this is from the vibration of the volume of air in the portion of the stomach undergoing relaxation secondary to the preceding contraction.

Leube attaches considerable importance to palpation of the stomach-tube, and the distance to which it descends; it is by no means easy to feel a soft rubber tube in the stomach through the abdominal wall, so his test is not at all reliable.

To Einhorn is due the credit of developing the method of trans-illumination of the stomach as an aid to diagnosis. In his hands, no doubt, it is valuable, but the clinician must learn to make his diagnosis by other means.

Fenton B. Turck, with remarkable ingenuity and untiring energy, has perfected the gyromele. He is not only able to determine the position and outlines of the stomach, but also the patulous condition of the pylorus; and he has lately shown a beautiful skiagraph with a duodenal sound in position. Probably the most valuable feature of the instrument is the readiness with which he obtains material directly from the wall of the stomach for chemical examination.

In our allusion to lavage, it has been as a preparatory measure for other methods of diagnosis, but the washing-out of the stomach is in itself one of the best methods of diagnosticating dilatation. By it we can determine the presence of food-stagnation, and upon this symptom alone Cohnheim bases his diagnosis of dilatation, while all authorities agree that it is an essential symptom. In atonic dilatation water flows into the stomach rapidly, making a whirlpool in the funnel; the outflow is very slow. This marked difference is not noticed in the early stages of dilatation from obstruction.

Differentiation of Atonic from Obstructive Dilatation:

Obstructive.	*Atonic.*
History.—Symptoms of ulcer, cancer, or some peripyloric neoplasm or disease.	Gastritis, malnutrition, wasting diseases, excessive indulgence in eating and drinking.

Presence of a Tumor.—Tumor usually found in the epigastrium.	Tumor absent.
Peristaltic Waves. — Pronounced in early stage.	Never present.
General Glandular Involvement.—Present in malignant stenosis.	Not found.
Cachexia.—Present in cancer.	Rapid emaciation and marasmus, but not cachexia.
Expression of Stomach-Contents.—By straining, the patient may expel stomach-contents through the tube.	Cannot be done in atonic dilatation.
Lavage. — Inflow and outflow are equally free.	Rapid inflow, delayed outflow.

Chemical and bacteriological tests are of no differential value.

DIFFERENTIAL DIAGNOSIS.—With due consideration of its general symptomatology and a careful application of all the test-methods of diagnosis, we are able to recognize the existence of dilatation, and there should be no confusion in the diagnosis. Gastroptosis, megalogastria, and dilated colon are the only reasonable chances of error. Food-stagnation and auscultatory percussion should eliminate these, yet Brown reports a case in which laparotomy was done, a thin-walled cyst evacuated, and about three pints of a viscid fluid drawn off; post mortem showed the cyst to be a dilated stomach, while the diagnosis had been intestinal obstruction. Other instances have been reported where dilated stomachs have been mistaken for ovarian cysts. The chances for mistaking a dilated stomach for some other condition are exceedingly small; our error lies in failing to recognize the existence of dilatation. In acute dilatation the diagnosis is at once established by passing the tube.

PROGNOSIS.—Atonic dilatation, if recognized sufficiently early, will yield to proper treatment. Acute dilatation, gastromesenteric ileus so-called, is a very fatal condition. Out of 212 cases collected by Laffer, in which the outcome was stated, 135 died and 77 recovered. The prognosis in obstructive dilatation depends upon the character of the obstructing cause. Broadly speaking, if the patient

is under forty years of age, we may hope that the cause is innocent; after that period, the probability of its malignant character increases. Non-malignant stenosis with dilatation is happily amenable to absolute cure. We may relieve for a long period, but may rarely cure when the cause is malignant.

CONTRAINDICATIONS TO OPERATION.—The presence of pronounced cachexia, general glandular involvement, metastatic growths, ascites, and anasarca precludes operative intervention.

TREATMENT.—Dilatation of the ordinary atonic type can be cured if recognized sufficiently early by rest, lavage, diet and medicinal measures.

Obstructive dilatation is a surgical condition, the operation varying with the pathology.

Acute dilatation of the post-operative type may be to some extent prevented by avoidance of handling of the stomach and intestines during operation, and by starvation for the first few days after operation and careful feeding later. We should be especially careful in handling cases in which some chronic dilatation is known to be present before operation. The treatment of this condition when it does occur is not in my opinion surgical. We must depend upon lavage and absolute stomach starvation. It requires nice discrimination to determine when it is safe to return to stomach feeding.

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FOREIGN BODIES IN THE STOMACH.

DEFINITION.—Substances alien to the stomach and not capable of being digested or passed, however introduced, constitute foreign bodies.

FREQUENCY.—While it is a possible accident that may occur at any time, it is a comparatively infrequent surgical condition. It is more the notoriety than aught else that gives to these cases their prominence.

Origin.—Foreign bodies are usually swallowed either by accident or design. It is a familiar performance of showmen to swallow knives, bars of lead, nails, tacks, pins, to eat broken glass, etc. Children by accident frequently swallow impassable foreign bodies. Lunatics sometimes indulge in this strange caprice. Imbecile old persons sometimes swallow their artificial dentures. It is by this channel that foreign bodies reach the stomach; yet true gastroliths are found in the stomach producing symptoms. Gall-stones may ulcerate into the stomach. Missiles may find lodgment in this cavity, as may appliances for anastomosis.

Detention in the Stomach.—The foreign body may be too large to pass the pyloric orifice, or its sharp points or angles may prevent its passage. Hair, threads, bits of grass or straw, and ground glass cling to the mucous folds of the stomach, become agglutinated by mucus, and, influenced by the perpetual motion of the stomach, are rolled into balls too large to be discharged.

A large brass fork has been retained in the stomach for two and one-half years; pocket knives have remained many months; hair-balls have gradually accumulated during three or four years. Upon the other hand, the swallowing of a foreign body has produced immediate symptoms demanding relief, or producing the death of the patient from obstruction or laceration.

SYMPTOMS.—As Credé remarks, the swallowing of indigestible articles is a frequent accident, but they are usually ejected or passed *per vias naturales.* Occasionally they produce adhesions of the stomach to the abdominal parietes, and are thus dis-

charged spontaneously; others remain in the stomach and give rise to no serious inconvenience for some time, ultimately, however, producing death by prolonged digestive disturbance or by perforation.

Dr. LeDentu reported a case to the Academy of Medicine, Paris, in which the man the night before had swallowed a wooden spoon 27 cm. long; some eight hours after swallowing the spoon he complained of a severe pain in the abdomen, and a sense of tearing in the region of the stomach. He was examined and the spoon distinctly felt through the walls of the stomach. Twelve hours after the accident he was given an anesthetic and prepared for operation; the spoon could not then be felt. On opening the abdomen, one end of the spoon was felt down in the pelvis, the other just below the umbilicus. The most minute examination could not discover the part of the stomach perforated by the spoon, yet the patient made a complete recovery. This case is related in detail to emphasize the possible behavior of long foreign bodies that may become transverse and subject to the muscular action of the stomach, producing immediate perforation. In 13 out of 90 cases collected by Friedenwall the foreign bodies produced abscesses.

In other cases vomiting, pain, and progressive emaciation supervene within a variable length of time. Cunningham and Warbase record cases of marked gastric tetany caused by the presence of foreign bodies in the stomach.

Berg reports a case of hair-ball in the stomach of a woman 26 years of age, who had been suffering with gastric symptoms for three years; for two years she had had a large epigastric tumor. Since childhood she had been in the habit of chewing hair. The mass when removed weighed 900 grms. These masses are called trichobezoars, and the case above mentioned is but one of an interesting series of cases collected by Dr. Nathan Jacobson, nineteen in all, to which he adds one of his own. His patient was a little girl eleven years of age, who had for some six months suffered with colicky pains, especially at night, situated in the left umbilical region; and there was frequent vomiting of a frothy mucus. A tumor was felt in the epigastrium, hard, nodular, and quite movable. The lower border reached to the umbilicus. No specific diagnosis was made, but exploratory operation showed

that the tumor was within the stomach. It was removed by gastrotomy, and the patient made a good recovery. The tumor was six inches in its longest diameter, in breadth two and a half inches; its contour presented a perfect cast of the stomach. "It is interesting to note," says he, "that the conical end passed into the duodenum, and that the pyloric prolongation meets the cardiac portion nearly at a right angle, and scarcely can be said to describe a curve." The diagnosis of these hairy masses in the stomach is practically impossible without a full history of the case, yet Caroline Pease made a correct diagnosis in one case, and boasts very vaingloriously over her remarkable acumen.

PHYSICAL SIGNS.—Long bodies, like spoons, knives, etc., may be palpated. Large globular masses may be felt, or, as in the above instance, a distinct tumor may be formed. Where nails, knives, and numerous bodies are in the stomach, we may elicit crepitus. The presence of a foreign body in the stomach with consequent irritation to its walls may result in adhesions and finally, perigastric abscess; this abscess is usually located over the anterior surface of the stomach. The X-rays will materially aid us in discovering and locating the foreign body.

COMPLICATIONS.—Aside from the immediate gastric phenomena and disturbances of digestion, many complications occur from the presence of a foreign body in the alimentary canal. Hemorrhage, local peritonitis with adhesions, abscess, intra-peritoneal perforation, and peritonitis may all occur while yet the body remains in the stomach. But its passage through the pylorus does not insure its exit from the body; anywhere in the intestinal canal, but especially at the ileo-cecal valve, it may become arrested and give rise to obstruction; or, in passing along the channel of the intestines, it may perforate their walls and set up a fatal peritonitis.

PROGNOSIS.—When a foreign body too large to pass the pylorus has been swallowed, the hope of its being discharged spontaneously by ulceration through protective adhesions is so impracticable as to be unworthy of consideration. The almost certainly fatal termination by one of the many complications is to be expected. Of the twenty cases of hair-balls in the stomach collected by Jacobson, ten were submitted to operation and all recovered. The simplicity and safety of gastrotomy place the operative mortality, if done promptly, at not over 3 per cent.

SURGICAL INDICATIONS.—If a foreign body has been swallowed, and it is too large to pass the pylorus, or if many small foreign bodies are recognized as detained in the stomach, *gastrotomy* should be done immediately.

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ULCER OF THE DUODENUM.

DEFINITION.—A necrobiotic process beginning in the mucosa of the duodenum, consequent upon impaired nutrition and autodigestion, with a tendency to involvement of the muscular and serous coats.

SYNONYMS.—Simple, peptic, round duodenal ulcer.

Frequency.—As compared with ulcer of the stomach, Krauss gives the statistics of Andral, 2 to 92; Trier, 1 to 93; Willigks, 1 to 38. This was thirty odd years ago.

It is still the custom of all writers to speak of the infrequency of duodenal ulcers. The revelations of surgery in the last few years have established the fact that duodenal ulcer is a comparatively frequent affection. The relative frequency of duodenal to gastric ulcer in this country is best established in all probability by Mayo's figures. In 1905 out of his last 231 cases of gastric and duodenal ulcer the duodenum was involved 74 times. From July 24, 1905, to March 23, 1907, he found 87 gastric ulcers, 98 duodenal, and 15 with separate involvement of the stomach and duodenum. This alone is evidence that the disease is common enough.

ETIOLOGY.—**Age:** It is found at all ages, even in infancy, but is most frequently met with from thirty to sixty years.

Sex: It is more common in men than in women, the ratio being about four to one (Fenwick). Mayo reports about 77 per cent. of his cases being in males, while Seymur Taylor gives the percentage as 72. Moynihan in 51 cases of duodenal ulcer found it in about 32 times in males and 19 in females.

The etiology of duodenal ulcer is analogous to that of gastric ulcer. The underlying predisposing cause is impaired nutrition, disturbed circulation in a definite area of the mucous membrane. This renders the part amenable to the corrosive action of the acid gastric juice. The upper two inches of the duodenum is to all physiological intents but a gastric extension. The stomach-contents reach this portion of the duodenum unaltered in their chemical characteristics, and therefore are capable of exerting their destructive effect upon the mucous membrane of lowered

resistance; and it is not until the acid chyme meets the pancreatic secretion that it is rendered alkaline and non-corrosive. In substantiation of this reasoning we find that duodenal ulcers are located above the common duct. When ulceration exists below the common duct, its etiology is the same as that of intestinal ulceration in general.

Collin found ulceration following burns in 38 instances out of 297 cases, and Porfick reported one instance in which ulceration of the duodenum developed eighteen hours after the occurrence of the burn. Older writers found it more frequently than later observers; the aseptic management of the burned surface has materially lessened this complication, proving conclusively that the duodenal ulceration was due to septic infarction. Its occurrence in erysipelas, frost-bites, and other remote infectious processes leaves but little reason to attribute to burns themselves any special potency other than sepsis in the etiology of duodenal ulcer. Perry and Shaw collected 21 cases illustrating the connection of duodenal ulceration with septic processes.

Practically all authors are in accord as to the etiology of duodenal ulcer. Bland Sutton is a striking exception; he stated before the London Clinical Society that duodenal ulceration had an etiology quite different from that of gastric ulcer; he believed that it was only found in alcoholics. In refutation of this statement it is only necessary to refer again to Collin's table; from it we see that duodenal ulceration was found 42 times in individuals under ten years of age, while several instances are reported of its occurrence in infancy. I have operated upon one case in a boy of seven years of age.

PATHOLOGY.—**Site**: The ulcer is usually situated on the anterior wall, and in the first portion of the duodenum. In a series of 262 cases found, Collin found that in 242 the ulcer was in the first portion of the duodenum. Mayo states that in 96 per cent. of his cases the ulcer came up to or within three quarters of an inch of the pyloric sphincter.

Number: Generally single; may be multiple.

Size: The size varies from a quarter of an inch to two inches —rarely more than a half inch—in diameter.

Character: Duodenal ulcer possesses practically the physical characteristics of gastric ulcer and is most frequently of the indurated type. 68 of 74 reported by Mayo were of this class.

The surrounding mucous membrane is healthy, but the muscular wall shows a limited round-celled infiltration. When healed, smooth cicatrization without restoration of the mucous membrane is found, contraction and partial stenosis resulting. In ulcers of the posterior wall adhesions to the pancreas are usual. Those on the free surfaces acquire attachments to the gall-bladder, colon, etc. Periduodenitis, constricting and distorting the biliary ducts, is a frequent operative observation.

In the progress of duodenal ulceration two complications arise, hemorrhage and perforation, which constitute a part of the pathology, but which will be considered under the complications.

SYMPTOMS.—The difficulties encountered in obtaining the clinical features of duodenal disease are readily comprehended if we appreciate its direct anatomical connection with other organs, its peculiar shape, its deep position, and its relations with other viscera. The possibility of diagnosticating duodenal ulcer is denied by physicians of the greatest acumen, yet, upon the other hand, ample evidence is produced showing that such diagnoses are made with exactness.

At the outset we are confronted with the alarming truth that duodenal ulceration may exist without prodromal symptoms, and may declare itself only by the occurrence of fatal hemorrhage or the development of general peritonitis consequent upon perforation. In Weir's list of fifty-one cases of perforating duodenal ulcer, twenty-five only had previous symptoms suggestive of duodenal disease. Schwartz says that in 20 out of 25 instances perforation occurred when the patient was in apparently good health. Clogg claims that one half of the cases of perforating duodenal ulcer give rise to no symptoms before perforation, while Smith reports five out of fourteen perforating ulcers without previous history. The frequency with which duodenal ulcer is latent has been explained on the grounds of the comparatively slight muscular effort required for that organ. However, it is worthy of consideration to know that duodenal ulceration occurs most frequently in men from the ages of thirty to sixty years, and that, while they may not show pronounced gastric symptoms, they bear the mark of ill health. Failure to elicit the existence of digestive disorders is frequently the fault of the investigator. Between the extremes of pronounced symptoms and those cases of absolute latency,

there is every shade of varying. Burwinkel, therefore, states it rather strongly when he says that in *all cases* there is a history of long dyspepsia, in which symptoms of ulcer appear at intervals.

Vomiting is not a constant nor persistent symptom, but acid eructations are common. When vomiting does occur, it is the acid chyme. It is usually not copious and rarely contains blood or bile.

Pain: Pain, a more frequent and valuable symptom than vomiting, according to Beckmann, was absent in only 16 out of 80 cases. The pain is described as located just below the gall-bladder, to the right of the median line. Buquoy assigns the pain to a zone corresponding to the inferior border of the liver, between the false ribs and iliac crest. The location of the pain is variable and not so important as its character and time of occurrence. It is often sudden, colicky in character, resembling attacks of gall-stone colic, radiating to other localities, upward especially; so intense may it be that convulsions will attend it. It occurs usually three or four hours after taking food. The time of the occurrence of pain has no immediate bearing upon the recognition of duodenal ulcer. If the pain does not occur until two to four hours after taking food, we may safely infer that the ulcer is in the pylorus or in the duodenum. When pain occurs immediately after taking food and then subsides to recur later, the inference is that we have both gastric and duodenal ulcer (Moynihan). While we cannot assign a definite cause for pain in duodenal ulcer, it is probably (according to Robson positively) due to the passage of the acid gastric juice, and while the stomach is concerned with the act of digestion the patients are comfortable. Therefore it is that they sometimes complain of a hunger pain. The pain resulting from one meal comes on just before the next and is relieved by taking food, an alkali or even fluid. It is said that solid food relieves the pain of duodenal ulcer and intensifies that of gastric ulcer. The pain of duodenal ulcer has not the tendency to radiate that we find in pain due to gallstone or to gastric ulcer. Vomiting frequently occurs in its acme, affording immediate relief. It is interesting to remark how quickly the patient returns to a normal condition after the subsidence of the pain. His appetite is good, the tongue clean, and he feels unusually well until a reoccurrence of this symptom, which may not be at any definite interval.

Tenderness: There is some tenderness in the region of the gall-bladder and below it, but a distinctive point of tenderness is not ascertainable. In duodenal ulcer neither the referred pain nor dorsal tenderness so characteristic of gastric ulcer is ever found.

Hemorrhage: The appearance of blood in the stools is perhaps the most constant and reliable of all symptoms of duodenal ulceration. The occurrence of hemorrhage is, unfortunately, only too frequently the first symptom. The blood in the stools may be fresh, but usually appears as melena. When melena continues for several days following symptoms of internal hemorrhage, syncope, acute anemia, etc., it points most significantly to duodenal ulcer as its origin. At the time of hemorrhage the patient may vomit reflexly, but the vomitus rarely contains blood. The amount of hemorrhage may be sufficient to cause immediate death. A careful examination of the stools after the method of Boas will show blood in fully 75 per cent. of cases. In fact, the disappearance of occult hemorrhage is used as a test of cure in both gastric and duodenal ulcers.

Jaundice: In frank cases in which there is frequent vomiting and pronounced pain, icterus may appear. It is a symptom especially, however, of those forms of duodenal ulcer of septic origin, in which the pathological condition is quite different from that of the simple round ulcer, the inflammatory edema producing the obstructive jaundice. Again, we may have jaundice in cicatrizing duodenal ulcer, the cicatrix directly obstructing or distorting the common duct. Periduodenitis secondary to ulceration not infrequently produces jaundice, confounding the differentiation from gall-stone.

So meager and obscure are the symptoms of duodenal ulcer that Weir, Kinnikutt, Ewald, in fact almost every writer, holds that operation or post-mortem examination is necessary for confirmation. Yet Burwinkel insists upon the simplicity of the diagnosis, which he bases upon five symptoms, and confirms it by five cases within five years. His patients were men approaching forty years of age, and his cardinal symptoms are: (1) melena, following symptoms of severe internal hemorrhage, continuing several days without vomiting; (2) the general condition; (3) the results of rectal palpation; (4) pain not appearing for two

or three hours after meals, not irradiating around the back, and localized near and above the umbilicus; (5) breath not fetid, tongue clean, appetite good.

COMPLICATIONS.—(1) **Hemorrhage:** The symptom hemorrhage is so conspicuous a feature of duodenal ulcer, and so frequently the cause of death that we must regard it as a complication. The bleeding is the result of erosion of large blood-vessels; pancreaticoduodenal was the source in 12 cases; Collin also found hemorrhage from the vena portæ twice, and from the aorta twice. In every case of enterorrhagia rectal examination should be made, the condition of heart, liver, and kidneys should be determined, and due attention should be given to the history before concluding that its origin is duodenal. In view of the great mortality from hemorrhage, if diagnosis is reasonably certain, operative intervention is certainly warranted; against this procedure might be urged the liability to secondary hemorrhage.

(2) **Perforation:** This is the most serious and frequent complication of duodenal ulcer. The frequency of perforation is given as 43 per cent. (Chvostek), 69 per cent. (Collin) and 50 per cent. (Robson). It can be judged from this that the risk from perforation is great. Ulcers on the anterior wall, because of their greater frequency and location, are especially liable to perforate, permitting escape of the contents of the viscus into the peritoneal cavity. The perforation is, of course, usually single, but Biggs reports a case of two ulcers with simultaneous perforation. The occurrence of perforation is marked by the onset of acute lancinating or burning pain; not infrequently the pain is felt in the iliac region, as has been emphasized by Dreschfeld. Fluid escaping from the duodenum or ruptured gall-bladder flows between the colon and liver, and thence directly downward on the outside of the ascending colon, reaching the iliac fossa (Weir). This may account for the localization of pain there. But in the great majority of instances the pain is in the right hypochondrium, occasionally in the epigastric or umbilical regions, but usually well to the right. The severity of the pain itself may produce marked depression, with nausea and vomiting.

Shock is not a necessary symptom. Weir says: "It is not often met with." Barker found severe shock present in about

one-half the cases. Immediately after the occurrence of the perforation symptoms of peritoneal irritation rapidly develop. Sensitiveness in the umbilical, hypochondriac and iliac regions, and general abdominal rigidity are present. Later, peritonitis supervenes, the rigidity yields, and we have diminished area of hepatic and splenic dulness, free peritoneal fluid, distended abdomen, absolute constipation, and weak pulse.

Such are the symptoms and signs at the time of rupture, clearly indicative of perforation-peritonitis, but by no means diagnostic of duodenal perforation. How then may we reach a diagnosis? Obscure as the course of the disease may be in many instances, yet there are previous symptoms present sufficiently often, if only they are obtained, materially to assist us. Weir attaches the greatest importance to the ***history,*** regarding it second only in value to the ***sharp onset of pain.*** Indeed, if we exclude the age and sex, dyspeptic symptoms, the occurrence of melena, and the attacks of pain in the region of the gall-bladder, with or without vomiting, when perforation does occur we have nothing but pain and shock—the latter symptom frequently absent—upon which to base a diagnosis. Yet, given a patient with or without a history, who is suddenly seized with this character of pain, evincing the general symptoms which Gübler has termed 'abdominal peritonism,' we have left to us exploratory incision, a step as essential to diagnosis as to treatment.

Differential Diagnosis of Duodenal Perforation.—The symptoms of duodenal perforation so closely resemble the fulminant form of perforative appendicitis that it is not surprising that errors are constantly being made, and with Richardson we must confess that the greater our knowledge of intra-abdominal pathology, the more lenient we become toward those who make errors in diagnosis. It must also be differentiated from perforating intestinal typhoid ulcer. Many symptoms set forth in writing appear common to all, yet to the experienced clinician there is an indescribable difference. However, it is possible to draw the distinction, and the leading differentia may be thus tabulated:

Perforated Duodenal Ulcer.	*Gangrenous or Perforative Appendicitis.*	*Perforated Intestinal Typhoid Ulcer.*
History.—Gastric dyspepsia, vomiting, and melena.	Gastric function not impaired. Previous attacks of pain and tenderness in iliac region common.	A knowledge of the typhoid state. A history of diarrhea, and may have prodromal symptoms of abdominal pain and local peritonitis.
Age.—From thirty to sixty years most frequently.	More common from ten to thirty years.	Usually in young adults, twenty to thirty years.
Sex.—Males, four to one.	Somewhat more common in lads, though frequent in both sexes.	More common in males, almost three to one.
Pain.—In the right hypochondriac, epigastric, and umbilical regions.	May be general, but greatest intensity in right iliac fossa.	Usually referred to the right iliac region.
Tenderness.—Rather extensive zone along the margin of ribs and in region of gall-bladder.	McBurney's definite point of tenderness.	Most persistent in lower zone of abdomen, in iliac and hypogastric regions.
Tumor.—Absent.	May palpate enlarged appendix; in some instances a distinct tumor.	Absent.
Peritonitis.—Extends from above downward.	Extends from below upward.	Quickly becomes general.

(3) **Stenosis of the duodenum** with consecutive dilatation of the stomach, the result of cicatrized duodenal ulcer, is a complication of very infrequent occurrence. Its general features would in no way differ from dilatation of the stomach due to pyloric obstruction, and they are considered under that subject.

DIFFERENTIAL DIAGNOSIS.—Analogous as we have found the two conditions, gastric and duodenal ulcer, to be, yet there are certain differential diagnostic points when present that will lead us to correct conclusions. It is essential for care to be taken in making the differentiation, as duodenal ulcer is much less amenable to medical treatment than is gastric ulcer. Then, too, the complications are more severe, hemorrhage being more rapidly fatal and perforation more frequent and insidious.

Gastric Ulcer:

Duodenal Ulcer.	*Gastric Ulcer.*
Age.—Forty years or after.	Most commonly under forty years.
Sex.—In men, four to one.	More frequent in young women.
Vomiting. — Not conspicuous, frequently absent.	A common symptom.
Time of Vomiting.—Two to four hours after taking food.	Promptly after eating, rarely longer than one hour.
Hematemesis.—Infrequent.	Frequently present, in almost every case at some time in its history.
Pain.—In the right hypochondrium or parasternal line.	In the epigastrium to the left of the median line, and in the left hypochondrium.
Time of Pain.—Two to four hours after eating.	Usually immediately after eating.
Radiation of Pain. — Usually downward, occasionally to the chest.	To the back, and to the left shoulder.
Tenderness. — Over the duodenum, near the region of the gall-bladder, diffused along the right costal arch.	Left epigastric triangle, and point-tenderness in the back to the left of the tenth to twelfth thoracic vertebræ.
Visible Blood in Stools.—Commonly present.	Only occasionally seen.
Jaundice. — Though an occasional, yet a valuable symptom.	Occurs only as a complication.

The more important differential points are the following: First, the pain in duodenal ulcer comes on late, frequently just before the next meal, and is relieved at times by taking food. The failure of the pain to radiate is also significant. There is more frequently present a visible amount of blood in the stools. The patient may have a good appetite and slight disturbance of his general health. Jaundice when present is a valuable symptom.

Gall-Stone Colic: The location of the intensity of the pain, the vomiting in the height of the paroxysm, and the occasional appearance of jaundice, in a clinical way make gall-stone colic and duodenal ulcer close kin, yet even here we may show that they are of different parentage.

Duodenal Ulcer.	*Gall-Stone.*
Pain. — Frequent attacks of pain after eating through a period of weeks, months or even years; it recurs at definite times and with some regularity.	Attacks are usually at night, at irregular intervals, not associated with irregularities in diet, and usually relieved in 24 to 48 hours by the passage or arrest of the stone.
Jaundice. — A rare symptom, usually transient.	Constant, if the stone is in the common duct.
Gall-Bladder.—Not enlarged.	Frequently enlarged.
Hepatic Enlargement.—Liver is usually small.	Liver usually large.
Stools.—Normal, or melena.	Clay-colored.
Vomitus.—May contain blood.	Never.

PROGNOSIS.—The detection of cicatrices in the duodenum at autopsy is undisputed proof that these ulcers do heal, yet so favorable a course is not to be hoped for so frequently as in ulcer of the stomach; and should it occur, stenosis from contraction is likely to develop. Unfortunately the tendency of duodenal ulcer is destructive, and while the disease may have a variable duration of from a few months to five years, yet it usually pursues a much more acute course, terminating suddenly in fatal hemorrhage or perforation.

TREATMENT.—Moynihan makes the positive statement that in duodenal ulcer surgical treatment should always be adopted. The outlook for the patient otherwise is hazardous. As with gastric ulcer the ideal operation is excision. Complete excision of the mobile portion of the duodenum has been recommended, but the anatomy of the organ does not lend itself readily to such a procedure. The danger of malignant changes at the site of an ulcer is nothing like so great in the duodenum as in the stomach. Gastrojejunostomy is even more satisfactory in its result in duodenal ulcer

than it is in gastric ulcer. For these reasons it will probably continue to be the operation of choice.

When perforation is diagnosed the abdomen should be opened as quickly as possible. As a rule, the ulcer will be found in the first two inches of the duodenum and on the anterior surface, and may be closed by a purse-string suture or by Lembert's sutures. Attention should be given to the toilet of the peritoneum. It should be cleansed by flushing if there is great extravasation. If only a limited field is soiled, wiping with gauze is all that is necessary. A suprapubic drain is much used when there has been extensive soiling of the peritoneum. Drainage of Morrison's pouch through the right lumbar region is added by some. Usually this is all that is required or should be attempted at the time. These patients are in such extreme condition from pain and shock that the operation must be completed as quickly as possible.

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CARCINOMA OF THE DUODENUM.

DEFINITION.—An atypical epithelial neoplasm, originating in the glands of Brunner or in the investing epithelium of the duodenal mucosa.

FREQUENCY.—Statistics from all sources show that primary malignant disease of the intestine affects the duodenum in about one per cent. of the cases. By a systematic analysis of the reports of all post-mortem examinations made in Guy's Hospital from 1826 to 1894, Perry and Shaw found that there were recorded thirteen cases of primary carcinoma of the duodenum, and nine of primary sarcoma. It is quite frequently, however, invaded by growths from neighboring structures, especially from the pancreas and bile-ducts. As we have shown, cancer of the pylorus rarely invades the duodenum; but Pye-Smith records a case in which the duodenum was secondarily involved from a cancer of the pylorus. Mayo has had three cases of primary cancer of the duodenum, one coming from ulcer, one possibly so and one of unknown origin. He considers the relationship unimportant. He has observed the development of cancerous changes in the edge of a duodenal ulcer that had extended into the stomach. This illustrates the marked preference of cancer for the stomach over the duodenum.

ETIOLOGY.—The obscurity which invests the origin of cancer wherever found obtains here, yet there are certain physical reasons which explain the localization of cancer in the duodenum upon the theory of irritation; they are its fixation, which renders it subject to injury; the change which takes place in the chemical reaction of its contents; and pressure, especially from an overloaded colon (Huelin).

Age: It is a disease of middle and advanced life.

PATHOLOGY.—Carcinoma of the duodenum is usually of the cylindrical-celled type. The growth may appear as a disc-like deposit, as an encephaloid mass, or in other cases it may implicate the circumference of the bowel, causing annular constriction. Its site may be above the opening of the common duct, surround-

ing it, or below it. The upper portion is its most usual location. The site of the growth is of material interest in its symptomatology. Whatever the shape or character of the growth, its tendency is to produce intestinal occlusion, and the changes consequent upon this affect very definitely the duodenum itself, the pylorus and stomach, and obstruct the outflow of bile, which initiates a new pathological process, materially complicating the course of the disease and confusing the diagnosis.

The tendency of duodenal cancer is to necrosis and ulceration, in which process large blood-vessels may be opened. Ulceration, which is superinduced by action of the gastric juice, affects usually the upper portion of the tumor.

SYMPTOMS.—It is not possible to indicate any one symptom or group of symptoms denotive of primary duodenal cancer. Secondary growths involving this part of the intestines are growths of neighboring organs, which may not organically affect the wall of the duodenum itself, but produce analogous symptoms. To every diagnosis, Whittier says, there should be appended the reserve clause, "an autopsy is needed before confirmation." The effect is to produce stenosis of the duodenum, wherever located.

Pic draws the following conclusions: The symptoms of this cancerous stenosis vary according to the height at which the stenosis is produced; a cancer above the papilla of Vater has symptoms almost identical with those of pyloric cancer; below the papilla it presents, in addition to these, a permanent reflux of bile and pancreatic juice into the stomach; a cancer around the papilla is a composite form. The diagnosis of cancer of the duodenum with biliary obstruction is much simpler than when the cancer is situated higher up, as the latter presents but slightly different symptoms from pyloric cancer.

There are certain symptoms, valuable in a general diagnostic way, of cancer of the duodenum, wherever located. The stenosis of the gut mechanically obstructs the flow of its contents, and we have a regurgitation through the pylorus into the stomach, giving rise to vomiting of partially digested food, mixed or not with bile and the pancreatic secretion, depending upon the site of the obstruction. As an inevitable result, also, of this obstruction, the extra muscular effort upon the proximal side

leads first to compensatory hypertrophy, then to conservative dilatation. The limited capacity of the duodenum necessarily reflects this upon the stomach, so that gastrectasia is, as a concomitant condition, a valuable sign of duodenal constriction.

The collapsed and flaccid state of the lower intestinal tract gives the abdomen an asymmetrical shape; on the right side and below, it is quite flat, while the left, that portion occupied by the dilated stomach, may at times show markedly prominent.

Pain is usually present; its location somewhat to the right, and its late occurrence after eating are valuable, but in no way differential. A tumor may or may not be found; comparatively large tumors are concealed by the proximal dilatation.

(a) **Cancers above the papilla** produce all the symptoms of pyloric obstruction. Although only slightly removed from the pylorus, yet this short distance gives some relief to gastric symptoms; patients have not the same disgust for food, disturbance does not appear so soon after eating, and thirst is not so great. In these respects only is the patient more comfortable. Hematemesis appears earlier and is more frequent. Melena will depend upon the patency of the constriction, since the bleeding usually takes place from the gastric side. Metastasis is earlier, cachexia more pronounced, and the progress more rapid. In pyloric cancer a palpable tumor is more likely to be found (Huelin).

(b) **Cancer at the Papilla**: These perivatrian cancers have not only the symptoms of occlusion of the gut itself, but in addition have the symptoms of biliary obstruction. Cholemia is usually pronounced, persistent and progressive. There is uniform enlargement of the liver, the gall-bladder becomes distended with bile, forming a distinct tumor. The patient usually has fever. The vomitus does not contain bile, nor do the stools show its presence; the urine becomes deeply stained. These symptoms, of course, vary with the absolute or relative constriction of the common duct. The progress of cancer in this locality, affecting, as it does, the important biliary functions, is very rapid. Descot and Beriel report an interesting case of perivatrian cancer of the duodenum, involving the common duct without causing icterus. The diagnosis was cancer of the pylorus. The autopsy showed the entire wall of the duodenum at the papilla, above and below it, involved; the bile-duct was rigid and dilated, and

through it the bile flowed uninterruptedly. Microscopical examination showed that it contained cancerous tissue.

(c) **Cancers below the Papilla:** As in the two preceding conditions, here, also, do we find the symptoms of intestinal occlusion with its gastric reflex. There may or may not be vomiting of blood. Melena may be present, but the important localizing feature is that the vomitus contains bile and pancreatic juice; this characteristic vomitus and the associate dilatation locate the obstruction below the papilla.

PROGNOSIS.—The prognosis is certainly and surely fatal. The duration of the disease is short, rarely longer than six months. It is interesting to note that Huelin, who, after Pic, has perhaps made the most thorough study of this subject, has discovered no case in which perforation took place into the peritoneal cavity. Several cases have terminated from hemorrhage. Death is usually due, however, to exhaustion and inanition or to biliary complications.

SURGICAL INDICATIONS.—Temporary relief only can be obtained by a gastrojejunostomy or, possibly, better still, jejunostomy.

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SARCOMA OF THE DUODENUM.

Sarcoma of the duodenum is an exceedingly rare condition. Perry and Shaw were able to find in Guy's Hospital Reports only nine recorded cases in sixty-eight years. The fixation of the duodenum gives to sarcoma in this locality a somewhat different symptomatic expression from that in other portions of the intestines. The obstructive symptoms may be as marked as in carcinoma. The distinguishing feature is the development of an immovable, rapidly-growing, distinctly-circumscribed tumor, attended by the most pronounced cachexia. The course of the disease leads to a rapid and certainly fatal termination.

ACUTE MECHANICAL INTESTINAL OBSTRUCTION.

DEFINITION.—By intestinal obstruction is meant an arrest of alimentation and digestion, an occlusion of the lumen of the bowel, irregular or arrested peristalsis, impeded or strangulated circulation.

CLASSIFICATION.—Intestinal obstruction is either mechanical or paralytic in its origin. Mechanical obstruction is caused: (1) By compression from without the bowel; (2) by obturation from within; or (3) as a result of structural changes in the wall of the intestine.

Clinically, mechanical obstruction is either acute or chronic.

GENERAL ETIOLOGY.—1. Mechanical obstruction of the bowel by compression from without may occur from a variety of causes, the most common of which are: through congenital apertures and into peritoneal fossæ, through mesenteric slits or diaphragmatic rents. These constitute the forms of internal hernia. Strangulation frequently occurs by congenital or adventitious bands, diverticula, and peritoneal adhesions. Compression may be due to twisting or torsion of the gut—volvulus; also to intussusception. Neoplasms and wandering viscera may likewise occlude the intestines by pressure.

2. Obstruction may occur from fecal impaction, foreign bodies swallowed, gall-stones, enteroliths, parasites, and from neoplasms growing into the calibre of the bowel.

3. Structural changes in the gut-wall itself not infrequently lead to obstruction. Cicatrical contraction occurring as a sequela of tubercular, typhoid, dysenteric, syphilitic, or malignant ulceration may so narrow the lumen of the bowel as completely to occlude it.

General Etiology of Intestinal Obstruction.

- From without by compression.
  - Through congenital apertures, into normal peritoneal fossæ, through mesenteric slits or diaphragmatic rents. Strangulation by congenital or adventitious bands, diverticula, and peritoneal adhesions. Twisting or torsion of the gut or its mesentery, volvulus, and intussusception. Neoplasms and wandering viscera.
- Obturation.
  - Fecal impaction, foreign bodies swallowed, gall-stones, enteroliths, parasites, etc.
- Structural changes in the walls of the intestines.
  - Cicatricial contraction as sequela of tubercular, typhoid, dysenteric, syphilitic, or malignant ulceration. Cysts and neoplasms growing into the caliber of the bowel.

General Pathology.—Intestinal obstruction, irrespective of its cause, is always followed by a series of consecutive pathological changes (Senn). Their order of development and intensity of action depend upon the cause; but certain destructive tendencies occur, irrespective of the character of the obstruction. In simple mechanical occlusion of the lumen of the bowel, retarding the passage of bowel-contents, a portion of the gut below the obstruction is empty and contracted. That above is distended with gas

and feces, circulatory changes develop from the compression of distention, and muscular paresis and infection from lowered resistance occur as certainly under these circumstances as they do in the most pronounced form of strangulation.

In chronic stenosis or obstruction, hypertrophy of the bowel-wall above the stricture is the natural result of excessive peristalsis and increased vascularity. This applies equally well to all parts of the alimentary canal, but the clinician is more familiar with it in the changes in the stomach incident to pyloric obstruction.

In all obstructions of a loop of bowel resulting in its strangulation, the affected portion becomes rapidly distended from the disturbance of circulation and the decomposition of intestinal contents. The gut loses its power of contraction, peristalsis is abolished, and the gross lesions are pronounced. The distended bowel is deeply congested, of a dark-red hue, and ecchymotic spots appear over its surface, evidencing the transudation from overdistended capillaries. The mucous membrane and underlying tissues become the seat of gangrenous inflammation. It is presumed that in the strangulated portion the circulation is brought to a stand-still; therefore, the subsequent changes which take place are brought about by the activity of microörganisms. "Intestines commonly contain various species of microbes, none of which are capable of producing disease under ordinary conditions. Two of the species, however, are known to be extremely powerful pathogenic agents when the tissues offer the peculiar conditions required for their growth. The first, the *bacillus of malignant edema,* finds in a portion of the intestine so strangulated almost ideal conditions for rapid growth. The second, *bacterium coli commune,* is extremely likely to invade the strangulated intestine" (R. M. Buchanan). It is by the action of these germs and the poison they elaborate that there is quickly developed in the strangulated loop progressive edema, multiple ulceration, gangrene, perforation, and septic peritonitis, conditions expressed by clinical phenomena which cannot be accounted for upon the ground of simple arrest of a function of the intestines, dejection.

In all forms of acute obstruction, but more especially in that from incarceration, the enlarged loop is filled with serous fluid.

This is largely a transudation, the result of venous stasis, and is not ingested matter. Transudation takes place from both the serous and mucous surfaces, and hence it is that we frequently find an effusion of free sanguinolent fluid in the abdomen. "And this ascites must not be overlooked in the examination" (Sarrazin).

The pathological effects of intestinal obstruction are far-reaching, involving at first a very limited area of the intestines, producing in a short time nervous and circulatory disturbances throughout the alimentary canal, invading the peritoneum, and affecting, if the patient lives long enough, the nutrition of all the organs of the body. The viscera, especially, are found dry and friable.

General Symptoms.—The anamnesis should receive due consideration in the symptomatology of ileus. The knowledge of a recent celiotomy or a severe contusion of the abdomen, the history of a perforated gastric or duodenal ulcer, would be invaluable evidence for the diagnosis of adynamic obstruction. Patients may recall incidents in their histories from which there may be surmised preëxisting peritoneal inflammations, and the logical sequence of bands and adhesions accounts for the present symptoms of strangulation. Attacks of diarrhea alternating with obstinate constipation, occurring in one of advanced years and declining health, excites at once the suspicion of malignant stricture. Intussusception, with its stealthy and indefinite symptoms, would often escape recognition, were we to disregard the predisposing conditions, the clinical history.

Weir tersely remarks, "The question is not what the cause is nor where the obstruction is, but is there an obstruction?" We accept the force of this statement as a plea for action. Yet on equally as high authority we know that ileus is but a "prevailing feature common to many different anatomical states," and a conscientious effort at a pathological diagnosis will protect us from surgical surprises and ante-mortem sections.

The most important feature of intestinal obstruction is the resisting power of the bowel-tract, and a very clear study of the effects of the disturbance or loss of this power has been made by Oser, of Vienna. Five important symptoms are by him attributed to disturbed resistance, and they occur in the following order:

(1) fluctuation of the intestine above the seat of stricture due to accumulated fluid; (2) increased and altered peristalsis, a symptom of much value and significance, especially in chronic obstruction; (3) vomiting, while generally not pathognomonic of obstruction, may under certain conditions become so; (4) meteorism; and (5) pain.

There are certain classical symptoms of intestinal obstruction which develop to a greater or less degree at some time in the progress of every case, regardless of the cause, and to these we must attach the greatest significance. They are pain, vomiting, collapse, absolute constipation, and meteorism.

Pain is one of the most common symptoms of intestinal obstruction. Its intensity, at the outset, depends upon the violence of the peristalsis, and the peculiar temperament or sensibility of the individual; subsequently, other factors come into play. Tension of the distended gut and injury to the peritoneum protract the suffering. In all cases of a progressive pathological process in which the obstruction is gradually brought about, the resisting power of the intestine accommodates itself to the approaching condition, and pain does not become a conspicuous diagnostic feature until absolute occlusion takes place. Quite the reverse of this occurs when from some cause there is sudden constriction or occlusion of the intestines. The offense, so to speak, precipitates violent and irregular peristalsis, and pain is one of the expressions of this perverted action; and hence it is that the pain of obstruction is usually spoken of as cramping, colicky, twisting, or binding. "In cases where the obstruction is complete, the pain is constant although liable to periodic exacerbations. In cases where obstruction is but partial, pain is distinctly intermittent" (Treves).

That peristalsis is one of the essential elements of pain is proved by the fact that obstruction in the large intestine usually excites but slight pain; whereas, in the small intestine, in which peristalsis is normally more active, occlusion or obstruction is followed by immediate and intense pain. Since Mr. Treves has pointed out the fact that the location of pain is no guide to the seat of obstruction, authors generally have contented themselves with emphasizing this statement. I feel warranted in accepting it this far, that the initial paroxysms of pain, which are largely

due to peristalsis, are usually reflected to the solar plexus, and hence referred to the umbilical region. But as the case progresses, when the engaged coil becomes distended with gases and fluid, and the peritoneum becomes involved, the pain then centralizes about the affected area. Coupling this symptom with the physical sign of local tenderness which usually develops at this stage, we may, in some instances, draw fairly correct conclusions as to the localization of the obstruction.

Vomiting is always an early and well-marked symptom of obstruction from all causes which bring about quick occlusion of the lumen of the gut, suddenly and sharply interrupting its normal peristalsis. From those chronic causes out of which gradually develop the final crisis of complete obstruction, vomiting may appear only as a late symptom. In certain forms, especially in intussusception, there may be an entire absence of vomiting. It is but a symptom dependent upon a number of causes, the chief one of which, however, is the upward axial current established by the descending peristaltic wave, so indisputably demonstrated by Mr. Brinton. It occurs first as a reflex nervous phenomenon, then as the result of increased intra-abdominal pressure from the over-distended and heavy-laden intestines, and from the irritation produced by the decomposition of the intestinal contents. Auto-infection and intoxication of the nerve-centers no doubt account for the later regurgitant vomiting, which persists after all peristalsis has ceased.

Clinically speaking, vomiting is a constant and most distressing symptom of acute obstruction. It appears early, is copious and severe; it usually begins by the eructation of gas, and singultus. The matters vomited are: first, the contents of the stomach, then bile, then brown or greenish material; this may continue for some hours or days with great retching, nausea, and distress before the matter ejected becomes feculent. "The sequence of gastric, bilious, and finally stercoraceous vomiting is perhaps the most important diagnostic feature of acute obstruction" (Osler).

Vomiting may appear as the initial symptom, preceding even the onset of pain; more frequently it appears later. If the stomach and intestines are full of fluid, vomiting will occur as an early and conspicuous symptom. A striking peculiarity of the vomiting in obstruction is that when it begins it usually con-

tinues to the last. It is not always attended with the distress generally ascribed to it; when large quantities of fluid are ejected without great effort, the patient oftentimes expresses marked relief, craves food, and frequently falls asleep; in a few hours there is a reaccumulation, and the distention oppresses him until he disgorges again. In the final stages of the disease vomiting may cease entirely.

Feculent matter is the decomposed contents of the jejunum and ileum; that the stercoraceous contents of the colon may regurgitate through the cecal valve, is perhaps true, but the putrid decomposition of the stagnant intestinal contents above this point is quite sufficient to give the vomitus its characteristic feculent odor and appearance. If the obstruction is high up in the jejunum or duodenum, vomiting appears early, is persistent, but never really stercoraceous. *Too much consequence must not be attached to this character of vomitus; it should not be relied upon for diagnosis; it is frequently absent or occurs late.* "It is certainly wrong to make fecal vomiting a plea for operation, and *vice versa*" (Greig Smith).

Roser's statement that the higher the seat of occlusion, the earlier and more violent the vomiting, generally applies.

Collapse: Vital depression inevitably occurs in every case of intestinal obstruction. It may not develop so rapidly as to warrant us in characterizing it as shock; it is only in acute obstruction that we have early, pronounced, sudden collapse so plainly manifested in the anxious expression, the sunken, pinched features, the cold, livid extremities, great restlessness, hurried thoracic respiration, fast, feeble, almost imperceptible pulse, and subnormal temperature.

The intensity of the shock and tendency to a rapidly fatal issue bear a certain relation to the suddenness and completeness of the strangulation, and to the presence of favorable conditions for the rapid absorption of poisonous substances produced in the strangulated part. It should therefore obtain that the greater the amount of bowel involved in the constriction, the greater the supply of poison and the more pronounced the shock. This statement is only relatively true, and is made with the full knowledge that profound shock sometimes characterizes the strangulation of a small knuckle, as for instance, in external hernia. The

injury to the peritoneum, the agonizing pain, and the sensibility of the individual all influence early shock. Profound collapse occurring late, that is, some hours or days after the initial symptoms, may be attributed to gangrene or perforation, with the rapid development of septic peritonitis and general intoxication. Death usually follows in from twelve to eighteen hours.

Constipation: A conspicuous feature in the clinical picture of obstruction is the absence of stools and flatus. The arrest of bowel-movement in mechanical ileus is due to the occlusion of the lumen of the gut at the seat of obstruction and to the paresis of the intestine below this point. Usually these two conditions appear coincidently, yet experience teaches that in some cases of strangulation flatus is passed after other marked symptoms are present. If sudden strangulation occurs high up in the intestinal tract, it is not inconceivable that the evolved gases of decomposition in the contents of the distal gut will cause flatus to pass audibly.

Constipation is not a prevailing symptom in all forms of obstruction. In paretic obstruction there is consecutive retention of feces, yet the use of enemata may succeed in causing the discharge of some feces and the free passage of flatus. So, likewise, in a number of the cases of intussusception the passage of fecal matter or gas is not completely arrested. Too much diagnostic significance should not be attached to constipation, for as Mr. Treves has said, "at most, it expresses a prevailing feature common to many different anatomical states."

Meteorism: It has been clearly shown by Kader, Murphy, and others that meteorism in any of the forms of intestinal obstruction is the result of disturbance to the circulation of the intestinal wall, and the decomposition and liquefaction of the intestinal contents. If a mesenteric vessel is occluded by embolism, or experimentally by ligature, paralysis, marked distention, and spots of gangrene on the convex side appear within forty-eight hours, thus proving that circulatory disturbances and the consecutive transudation are important elements in producing meteorism. The quantity of the ingesta is not an important factor in producing the distention. Murphy has clearly shown that the contents of a strangulated loop consist largely of a transudation from the mucous membrane due to the stagnant circulation.

The degree of distention depends upon the completeness of the obstruction, the duration of it, and the position of the obstruction in relation to the length of the bowel (Wyllie).

Meteorism is only present in that portion of the bowel above the seat of obstruction. The nearer the anus the obstruction the greater the tumidity, "a law which is the reverse of that applied to vomiting" (Wyllie). The site and development of the tumidity are valuable guides to the location of the obstruction. If the trouble is high up, that is, in the duodenum or in the beginning of the jejunum, the meteorism is confined to the epigastrium. If in the lower colon, we have at first a circumscribed area of tympanites, which soon becomes general. The manner of the development of the meteorism has some diagnostic value; in obstruction by strangulation it develops passively, so to speak, without perceptible peristalsis; in chronic stenosis the meteorism is accompanied by violent peristalsis, both seen and felt. The distinctness with which the bowel may be seen in rigid spasm depends upon the thinness of the abdominal parietes, and upon the amount of hypertrophy of the intestinal muscles, consequent upon the length of time there has been increased resistance to normal peristalsis.

The greatest importance must be attached to the five symptoms considered, viz., *Pain, Vomiting, Collapse, Constipation,* and *Meteorism.* Yet in the diagnosis of obstruction we must take into account every feature presented. In sudden strangulation, characterized by agonizing pain, persistent vomiting and profound collapse, the pulse is frequent, soft, and thready. In many cases, however, the pulse remains slow until affected by peritonitis from infection. I would emphasize the differential diagnostic value of the low temperature, subnormal in shock, and rarely rising above 100°, frequently remaining normal even after perforation and peritonitis. The respiration is influenced by the amount of distention and the presence of peritonitis. The anxious, oppressed look, dry tongue, offensive breath, rapid emaciation, great thirst, scanty urine, sometimes albuminous, all betoken the gravity of the case.

SEAT OF OBSTRUCTION.—The accuracy with which we can determine the seat of occlusion depends more upon the cause producing the obstruction and the history that attends its development

than upon any special symptoms arising therefrom. Occlusion of the colon may begin quite acutely, as in volvulus of the sigmoid, with some fixed pain, and a localized area of tympany, yet there is likely to be but little shock, and vomiting occurs late, or not at all. Further symptoms develop slowly. The progressively ascending meteorism, which soon becomes general and excessive, is quite characteristic of obstruction in this locality. Very different symptoms attend obstruction of the duodenum or jejunum; here pain is agonizing, collapse pronounced, vomiting ceaseless, there is only epigastric distention, and this may be relieved after vomiting or eructation of gases. Between these two extremes, the duodenum and colon, there are twenty-odd feet of doubt, and we can only in a general way locate the site of obstruction.

Without attempting to discuss the many anatomical and physiological reasons which influence the symptoms, it is clear, in the language of Mr. Treves, that "the small intestine is active and vigorously concerned in the business of organism," and the function of the large intestine is to a great extent passive; so will the symptoms be, relatively speaking, active or passive according to the part involved.

The large intestine is the most frequent seat of slowly developing stenosis, and if to our knowledge that cancer is the common cause we add the testimony of the age of the patient, the manifest cachexia, and the probable presence of a tumor, we are generally able to locate the disease. A history of peri-appendicular inflammation preceding an acute attack, characterized by the symptoms of gut-strangulation, would naturally guide us to search for a peritoneal band, and the ileum as the seat. Operation may disclose an entangled sigmoid or jejunum, yet, owing to the disposition of a given part of the small intestine to change its location, we are unable to fix any definite topography of this part of the alimentary canal.

In our efforts to locate obstruction in the lower bowel above the rectum, very little reliance can be placed in the quantity of enemata retained or in the conclusions derived from attempts to pass a tube beyond the sigmoid; and so far as my observation goes, little light can be obtained by speculum-examination after Kelly's method. Much has been written upon the function of the kidneys as influenced by the seat of obstruction; a marked in-

crease in the indican of the urine has been found by Jaffé when the occlusion was in the small intestine. This occurs too frequently in intra-abdominal disease, other than obstruction, to be of material diagnostic value. It is also stated that the nearer the stomach the obstruction the less the amount of urine secreted. This is faulty reasoning. It is true that in strangulation of the small intestine the profound shock, copious vomiting, violent pain, and trauma close to the abdominal nerve-centers, are all conditions which greatly lower blood-pressure and suspend the function of every organ. The oliguria is, therefore, more pronounced in high obstruction, yet it must not be made a point of diagnostic value. I have seen suppression for eighteen hours, following strangulation of the sigmoid by a band after celiotomy for removal of a diseased uterine appendage.

Wyllie attaches the greatest importance to the study of the patterns presented by the bowel in spasm. In chronic obstruction the hypertrophied and ectatic bowel in its violent peristalsis should be carefully observed, and in connection with other symptoms will aid us in determining the seat of obstruction. Yet I have seen the coils stand out individually visible and palpable, and upon operation I was entirely misled as to the seat of obstruction.

General Treatment.—There is but one measure that can be accurately and intelligently applied in the treatment of acute mechanical intestinal obstruction, and that is abdominal section. Non-operative treatment has at times resulted in recovery, the recovery, of course, being open to suspicion of being spontaneous. A knowledge of the pathology of obstruction makes very clear the utter hopelessness of accomplishing anything with medicine. The rapid progress of most cases to a destructive termination makes any hesitation in advising and applying surgical treatment, once the diagnosis is made, dangerous indeed. So urgent is the need for early mechanical relief in these cases that we should unhesitatingly advise early exploration in suspicious cases without waiting for positive evidence.

The seriousness of operation is in inverse ratio to the promptness with which it is undertaken. We must not only be early enough to anticipate, if possible, destructive changes in the involved bowel, but also to prevent the development of peritonitis from the passage

of bacteria through the still intact bowel wall under pressure of accumulated material in its lumen. If this is done, operation is robbed of most of its danger, though mechanical obstruction must always carry a serious risk on account of the primary pathology itself in many cases.

As Treves says, the most serious feature of all in the management of acute intestinal obstruction is delay, but prompt operation does not make it necessarily a simple affair. Intestinal resection may be required before the obstruction can be relieved, and not infrequently the condition will be found inoperable so far as removal of the primary pathology is concerned.

Without going into the details of technique there are some general considerations that we may take up here. First of all, gastric lavage should be done before operation. It is a distinct advantage to both patient and anæsthetist and greatly lessens the risk of aspiration pneumonia. Ether should always be used when a general anæsthetic is indicated. The incision should be made in the median line beginning just above the pubes and should be of ample length. In exceptional instances other incisions will be required, but this meets the indications in the majority of instances. Evisceration is less dangerous than the manipulation of the distended bowel within the cavity. The flaccid bowel below the obstruction rather than the distended coils above should be handled, if possible, in searching for the point of constriction.

When the involved bowel does not at once present itself, upon opening the abdomen, we should locate the cæcum. If this organ is distended, we must search the large intestine for the obstruction. If it is flaccid, we take up the flaccid ileum and follow it to the constriction.

If there is much accumulation in the bowel above the obstruction, we must evacuate the bowel contents by enterotomy, as it is highly septic and will be rapidly absorbed by the flaccid bowel below if allowed to pass into it. This is not so essential when the obstruction is quite low, if it is possible to return the intestine and close the parietal wound without too much tension. When resection is necessary and anastomosis is indicated, this last should be done by suture and the sutures should be tied with the utmost care to secure mere approximation of surfaces. The bowel is more or less œdematous and friable, and will readily necrose under constriction. On

account of the friability of the bowel, the propriety of making a temporary enterostomy instead of doing an anastomosis is a question that will frequently come up. When in doubt it is best not to attempt complete surgery, particularly when the obstruction is in the large intestine or quite low in the small. Dread of the care of an artificial anus and of a secondary operation for its relief should not make us hesitate when it offers greater hope of saving the patient's life.

In desperate cases that have been neglected the performance of enterostomy, upon the first coil of the intestine that presents in a parietal wound made under cocaine anæsthesia, is a blind unsurgical procedure, but will occasionally save a life.

Let us enumerate once more the causes of acute mechanical obstruction:

(1) Strangulation by bands.
   - *a.* Peritoneal false ligaments.
   - *b.* Meckel's diverticulum.
   - *c.* Omental bands.
   - *d.* Appendix vermiformis cæci.
   - *e.* Fallopian tubes.
   - *f.* Internal herniæ.
   - *g.* Slits in mesentery, omentum, peritoneal ligaments and membranous adhesions.
   - *h.* Kinks and twists due to peritoneal adhesions.

(2) Volvulus.

(3) Invagination.

(4) Stricture.
   - *a.* Benign.
   - *b.* Malignant.

(5) Obturation.
   - *a.* Fecal impaction.
   - *b.* Foreign bodies.
   - *c.* Gall-stones.
   - *d.* Enteroliths.
   - *e.* Parasites.
   - *f.* Mucous casts.

(6) Neoplasms.
   - *a.* Without the bowel.
   - *b.* Within the bowel.

Of these causes internal herniæ, neoplasms, and cicatricial contractions present special and characteristic features and will be considered elsewhere.

The consideration of the other conditions we will now take up somewhat in detail. The various forms of obstruction resulting from obturation present features in common. Gall-stone obstruction is dealt with at length as a type of this form.

Intestinal Strangulation by Bands.

Definition.—We group under this head those cases of intestinal obstruction attributable to bands, congenital or adventitious; peritoneal adhesions; vitelline remains; abnormally attached viscera; mesenteric slits.

Frequency.—Among 1,000 deaths from intestinal obstruction, Mr. Treves finds that 250 were due to strangulation by bands and through apertures. The frequency with which peritoneal adhesions and bands cause intestinal strangulation is variously estimated from 10 to 35 per cent. Statistics gathered in the last score of years, the days of peritoneal invasion, will no doubt place this cause well in front as an etiological factor.

Etiology.—It is a common post-mortem observation to find bands, cords, belts, and veils traversing various portions of the abdominal cavity, unmistakable evidence of preëxisting localized adhesive peritonitis, the result of appendicitis, peri-uterine inflammations, mesenteric adenitis, cholecystitis, and post-operative sequelæ. The formation of adventitious adhesions is readily comprehended. Following an attack of plastic peritonitis, soft adhesions of adjacent peritoneal surfaces form; if resolution does not speedily take place this may unite neighboring coils of intestines to each other, to the parietes, or to a solid viscus. Opposed peritoneal surfaces may be so anchored that the subsequent mobility due to exercise and function draws and pulls upon the adhesions, narrowing, lengthening, and rolling them into cords and bands, which finally become tough and unyielding (Ross). These peculiar bridges may be congenital abnormalities; or equally dangerous and identical mechanical conditions may be due to slender viscera abnormally attached, or to the diverticulum of Meckel.

These abnormal attachments may ensnare and strangulate the

intestine in many ways. As some one has said, it is curious to observe the extraordinary ingenuity exercised by the bowel in finding various ways to develop an obstruction. Whether around, over, or under is a matter of anatomical interest only. Some violent effort may precipitate the changed relation, just as an external hernia may appear after exertion, and immediate strangulation develop; or the constriction may be only partial at first, the subsequent changes in the incarcerated loop tightening the constricting cord to absolute strangulation of the gut.

Age: Strangulation by bands is more common in young adults, is rarely met with in advanced life; the author, however, encountered it in a man sixty-three years old.

Sex: Statistics prior to 1890 show this form of obstruction to be somewhat more frequent in males. It is quite possible that later records may show a relatively equal proportion between the sexes.

PATHOLOGY.—The ileum is the portion of the intestine usually involved in this form of obstruction, though, of course, no part of the small intestine enjoys immunity, and the flexures of the colon and cecum may become strangulated. The amount of bowel embraced may be only a small knuckle or several feet. If the constriction is only moderately tight at first, the violent peristalsis excited drags more and more bowel with its mesentery into the blockade. The extreme traction upon the mesentery, and its constriction are chiefly accountable for the rapid changes that occur.

The local and general pathological changes consequent upon strangulation have been thoroughly considered under the head of general pathology.

SYMPTOMS.—If we merely assume that there is intestinal strangulation without an effort to investigate its probable cause and location, we give but little encouragement to the cultivation of diagnostic acumen. In 68 per cent. of these cases there is a history of previous attacks of peritonitis (Treves). Carefully-taken clinical histories will reveal previous intra-abdominal troubles sufficiently often to reward the investigator. It was perhaps an appendicitis, tubal or ovarian inflammation, a gastric or duodenal ulcer, typhoid fever with deep but not perforating ulceration. Nor must we neglect to refresh the memory of a blow or

injury to the abdomen, followed by a few days of pain and tenderness and then forgotten. If the history is blank to all this line of inquiry the surgeon may assume with some degree of reason that the constricting cause is perchance a hernia through the foramen of Winslow or into one of the peritoneal fossæ, intrasigmoid, retrocecal, or duodenojejunal; or it may be due to Meckel's diverticulum. If the patient presents congenital defects the probability of the latter cause would be strengthened. It is needless to argue against the inestimable advantage of the consideration of these possibilities if operation is contemplated.

Mode of Onset: The symptoms of strangulation are usually violent and sudden in the onset, without pronounced warning.

Pain: The patient is suddenly seized with acute abdominal pain variously described as griping, twisting, or colicky. It is always severe, even agonizing. The colicky waves or paroxysms, the griping, is but an expression of the violent contortions of the intestine to overcome the resistance to normal peristalsis. It is thoroughly established by Mr. Treves as a clinical fact, that the more absolute the obstruction the less intermittency to the pain; hence, in the onset the pain may be distinctly paroxysmal, but as the halter tightens, the pain becomes persistent and continuous.

The seat of pain in this form of obstruction is no guide to the seat of obstruction; as the ileum is the part usually involved, the impression is conducted through the superior mesenteric plexus to the solar plexus, and from this center it is recognized. Therefore, in this form of obstruction the pain is referred, and is located by the patient about or a little above the umbilicus. A notable exception to this rule I observed and reported at the meeting of the Southern Surgical and Gynecological Association, 1894. In this case the pain and tenderness were felt at McBurney's point, and upon this misleading sign perforative appendicitis was diagnosed. At operation a false ligament was found passing from the bowel to the mesentery near the center of its vertical attachment. Beneath this several feet of ileum had become strangulated. While pain is a conspicuous and constant symptom of strangulation, it may be modified greatly by the temperament of the individual. The sudden cessation of pain can only mean the spontaneous release of the strangulated gut or impending dissolution.

Vomiting: Early vomiting is a reflex or nervous symptom, and its occurrence as an initial symptom depends largely upon the amount of food in the stomach. Soon after strangulation is effected other conditions act their part. The reverse current of anti-peristalsis, the distention of the intestines, the rapid accumulation and decomposition of the contents of the bowel, its noxious and irritating effect directly upon the mucous membrane of the intestines and stomach, toxemia, and peritonitis, are causative factors. Vomiting may come on with, or even precede pain; it is justly regarded as a constant and conspicuous symptom in strangulation by bands. Its occurrence does not materially aid us in locating the obstruction further than the recognized general law, the nearer the stomach the obstruction, the earlier and more persistent the vomiting.

The character of vomitus is: first, the contents of the stomach; then, bilious matter; later, brown or chocolate-colored fluid. The vomiting of feculent or stercoraceous matter, which is the only pathognomonic vomit of obstruction, does not usually occur earlier than the fourth day. A patient may die without the appearance of this characteristic vomit. Of course, it does not occur at all in high jejunal or duodenal obstruction. The persistent, ceaseless vomiting, accompanied by violent retching, is one of the most distressing symptoms in the early days of strangulation. Later, when the sensibilities are blunted and the act of disgorging is purely a mechanical one, the patient often expresses the greatest relief; the regurgitant effortless vomiting is a striking feature of the closing scene of intestinal obstruction.

Constipation: From the inception of the accident there is absolute constipation. All the bowel to the distal side of the obstruction, if not in a state of positive paresis, is relaxed and inert; consequently there is no bowel-movement of either feces or gas. It is well to bear in mind that some fecal matter, the contents of the lower bowel, may escape after high injections, and that a nutrient enema, coagulated and formed, closely resembling fecal matter, may mislead the attendants.

Shock or Collapse: Second only in diagnostic importance to the especially conspicuous symptoms, pain, vomiting, and constipation, is the general condition in acute strangulation of the intestines. The patient's general expression soon bears evidence

of the gravity of the condition. From the first he "looks ill," mental anxiety and physical distress are often characteristically depicted in the so-called "facies abdominalis." Early in the history of strangulation the signs of profound shock, even to collapse, develop: the pulse is weak and frequent; temperature rises not more than a degree from normal, is frequently subnormal; respiration is hurried and shallow; extremities are cold and livid; cold perspiration in great beads stands out upon the brow. There is great thirst and restlessness.

These symptoms, grouped by Gübler under the term "abdominal peritonism," are generally pronounced. Yet it must not be understood that the advent of every case is so stormy and terrible. I have seen complete strangulation for five days, the gut almost severed by the constricting bands, and yet the patient's pulse and temperature had varied but slightly from the normal. The intensity of the shock depends much upon the sensitiveness of the patient and resistance to infection, yet sooner or later in every case of intestinal obstruction the inevitable collapse appears. If early, it may be largely attributable to injury to the mesenteric nerves and to their direct connection with the pneumogastric. We could not in any other way rationally account for the great impression upon the circulation and respiration. Collapse occurring late, that is, after the general symptoms of obstruction have existed for some days, points only too surely to perforation, gangrene, or the advent of general sepsis.

The rapid emaciation and extreme prostration that afflict these patients with acute strangulation is erroneously attributed to the rejection of nourishment only. We must keep in mind known pathological facts. In intestinal obstruction there is a great drying-out process rapidly going on in the body, affecting naturally, at first, the viscera of the abdomen. They are dry not only because they are deprived of fluid for absorption, but there is free transudation of serum into the intestine on the proximal side of the obstruction, and this is constantly vomited, and represents so much water withdrawn. The absorption of poisonous products, evolved by the organisms now in possession of the tissues of low resistance, is perhaps the most potent cause of the great vital depression.

We would naturally expect to find, with the condition above

described, a marked diminution of urine; and there is an increased amount of indican, if the strangulation is in the small intestine.

PHYSICAL SIGNS.—**Inspection:** Assuming that the obstruction, except in very rare instances, is in the jejunum or ileum, there is but slight meteorism developed. The constant vomiting and eructation of gas, as Mr. Treves explains, serve to relieve in a great measure the distention. When, however, several feet of intestine are ensnared, then an asymmetrical distention may appear very early. When peristalsis is subdued and peritonitis develops, the abdomen becomes distended, noticeable at first in the epigastric region, later becoming general; but it is unusual for meteorism to attain any conspicuous degree in this form of obstruction. In very thin subjects the waves of peristalsis and patterns of intestinal coils have been observed.

Palpation: The abdominal wall is flaccid. At first there is some general hyperesthesia, but no distinct spot of tenderness until local peritonitis supervenes; in that area the muscles are resisting and tenderness is marked. We cannot outline any distinct tumor, hardness, or induration. Muscular rigidity appears as a sign in many clinical records of cases of sudden strangulation; I have not observed persistent rigidity; the muscles contract during paroxysms of pain, then immediately relax.

Percussion: In the first few hours percussion is practically negative. Soon moderate tympany appears. From the strangulated intestine and the adjacent irritated peritoneum there is poured out into the peritoneal cavity sero-hemorrhagic fluid; this free peritoneal fluid is detected early by dulness in the loins, and it rapidly increases. Appearing, as it does, before marked meteorism, it is easily detected; and if the patient is examined frequently, its increase hour by hour may be noted. Carl Boyer regards the "acuteness and rapidity" of the effusion as diagnostic of strangulation. In a patient of Dr. J. T. Altman, of Nashville, upon whom I recently operated, strangulation occurred at 2 a.m.; at 12 m., ten hours later, free fluid was first detected; at 6 p.m. it had markedly increased. I operated at 10 p.m. and found at least two quarts of bloody serum in the cavity. The strangulation was due to the appendix encircling the ileum at its cecal junction.

When a loop of intestine is incarcerated it becomes distended with fluid, and may in this way give rise to local dulness. In one such instance the suddenly-developed tumor, the well-defined dulness, and the general symptoms resembled an ovarian cyst with torsion of the pedicle. Had the patient been a female I am sure I would have made a mistake in diagnosis.

Auscultation: Auscultation gives no information except in cases far advanced, with large accumulation of fluid in the incarcerated coil; then we may hear tinkling or gurgling, or, if the patient will permit the necessary force, we may elicit clapotage.

DIFFERENTIAL DIAGNOSIS.—The mode of onset and early symptoms of intestinal strangulation by bands resemble many acute intra-abdominal conditions, in all of which the same general surgical indication, laparotomy, obtains. Yet precision in diagnosis contributes materially to the perfection of the technic and to correctness of prognosis.

Gastric or Intestinal Perforation: The symptoms that attend the occurrence of gastric or intestinal perforation are those of *abdominal peritonism,* so conspicuous in intestinal strangulation. Upon the history of the patient we must largely rely for guidance. An anamnesis of gastric, duodenal, or intestinal ulceration will direct further investigation to the seat of disorder. If perforation occurs from latent ulceration, in which all preëxisting symptoms are wanting, the difficulties of diagnosis are increased. Yet, even then, closely applied tests may solve the problem. Localized tenderness is usually well-marked in perforation; muscular rigidity, persistent, either localized or general, is rarely absent; abdominal distention with effacement of hepatic dulness develops early; vomiting attends the actual occurrence of perforation usually; after that it may cease—at least, be an inconspicuous symptom until the advent of peritonitis. In all forms of perforation feces and flatus may be discharged until bowel-action is inhibited by peritonitis. Hematemesis or melena would be of infinite diagnostic value.

Perforative or Gangrenous Appendicitis: Secondary, as these conditions usually are, to preëxisting disease of the appendix, the knowledge of previous attacks directs our attention to the appendix. The discrimination in the intensity and character of pain, the frequency of vomiting, and the degree of shock, are too fine

to rest a diagnosis upon; but the localization of the pain, the point-tenderness, and the muscular rigidity, are signs rarely assumed by intestinal obstruction. Temperature, so much discredited as a diagnostic symptom of appendicitis, is, nevertheless, of greatest differential diagnostic value in distinguishing appendicitis in the first twenty-four hours from intestinal strangulation; a fever of over 101° within this time practically precludes intestinal strangulation.

Biliary and **Renal Colic**: These merely require mention for differentiation. The character and duration of the attacks, and the functional disorders that attend them should eliminate them.

PROGNOSIS.—Death inevitably occurs in from three to seven days without operation. The patient succumbs to exhaustion or to peritonitis from gangrene and perforation. The probability of spontaneous release of the strangulated bowel is too small for consideration.

TREATMENT.—In the majority of cases of intestinal strangulation, if operation is done early, the procedure is simple enough once the site of obstruction is located. Often nothing more is required for the relief of the condition than the division of a band by tearing or cutting. If the band is vascular (omental bands, for instance), it will have to be ligated as well. If a patent Meckel's diverticulum or the appendix is found to be the cause of the obstruction it must be removed and the opening in the bowel closed by purse-string or other suture. So too an adherent Fallopian tube may have to be excised. In strangulation in mesenteric and similar slits some difficulty will be encountered in releasing the bowel, and the operation may be quite complicated. In obstruction by kinking due to adhesions, the operative procedure is usually simple, though at times complete separation of the attached surfaces is difficult or even impossible. Adhesions between adjacent coils of intestines are usually of less importance than those binding the bowel to other viscera or to the parietes.

When the integrity of the bowel has been impaired as the result of strangulation, resection followed by anastomosis or enterostomy must be done according to the demands of the case.

VOLVULUS.

DEFINITION.—Volvulus or twisting of the intestines embraces several different conditions. All the complicated forms of knotting and intertwining of the intestines so carefully unraveled and described by Leischtenstern, and by Küttner, are but forms of volvulus. So, likewise, we embrace under this head kinks of the bowel, and twisting of the bowel upon itself. The usual form of volvulus met with clinically is that in which the intestine has rotated on its mesenteric axis, the torsion and constriction affecting the mesentery, but not at first necessarily occluding the lumen of the bowel.

FREQUENCY.—Volvulus is comparatively an infrequent form of intestinal obstruction.

ETIOLOGY.—Kinking may occur at any part of the alimentary canal provided with mesentery, but as this accident is so unusual and rarely seen except in operations upon the intestine itself, we shall not here give it further consideration. True volvulus can only occur in that part of the intestine with long mesenteric attachments; consequently it is met with most frequently in the sigmoid flexure of the colon or in the ileum. Congenital malpositions, which are almost always due to unnaturally long mesenteries, are accompanied by a corresponding mobility of the bowel. A very accurate and scientific thesis by Curschmann upon the "Topography of the Alimentary Canal" gives many beautiful illustrations of abnormalities. A study of his observations has greatly elucidated the question of volvulus, and I can no longer believe that chronic constipation alone is a sufficient cause.

The underlying, predisposing cause is a congenital condition of elongated mesentery with misplaced and redundant loops of the bowel, to which abnormality we may add the torpidity and constipation habitual to those of advanced age. The ultimate crisis is no doubt precipitated by clogged intestinal contents, which produces pressure and tension.

While the sigmoid flexure is the classical seat of volvulus, other portions of the intestines may be so affected. Curschmann has found, out of abundant material, only one case of volvulus of the ascending colon. Treves was able to find only one, that reported by Mr. Curling. The cecum, whether congenitally elongated or enjoying unnatural mobility because of the unusual

length of its mesocolon, may, in bending over upon itself, become obstructed. Two cases of fatal obstruction in this locality are recorded by Curschmann. Twisting of the transverse colon is relatively rarer than other portions of the large intestine, "as the peduncles of the loop are further removed from each other at their base." It is in the descending colon and sigmoid flexure that we find the greatest deviation from the normal. "In early life enlargement of the sigmoid flexure, a sort of loop-formation, seems to be physiological. This infantile condition disappears in most individuals in later life, but autopsies have shown that in a certain number of adults this visible enlargement has persisted throughout life. The writer has found it in 15 out of 233 necropsies. In the 15 cases mentioned the length varied between 195 and 280 c.m., rather large figures when we consider that the average length of the large intestine in both men and women is 142 c.m. The sigmoid flexure alone (in three cases) was 110 c.m. long, as long as the measurement of the entire colon in a medium-sized adult" (Curschmann).

In the abnormalities here described the requisite conditions for volvulus are present. The mesentery is long and narrow, the ends are close together, and the loop of intestine of great length. There is ever present in such abnormalities more or less impediment to the passage of feces; and, as Grawitz, quoted by Senn, asserts, an important cause of volvulus is found to be an accumulation of intestinal contents above the constricted portion of the bowel, when it is easy to understand how it will rotate on its own axis. This tendency of the inflated bowel above the constriction to rotate around the point of constriction has been referred to in the etiology of Strangulation by Bands. The tendency of all mechanical laws is to continue the rotation. Rarely do we find unquestioned clinical record of a volvulus righting itself.

Age: It is met with most frequently in persons of advanced age, but this statement must not be interpreted to exclude the young. Numerous cases are recorded of its occurrence in early life.

Sex: It is far more common in males than in females; the ratio, according to Treves, is four to one.

PATHOLOGY.—The lumen of the gut is closed; the constriction

and strangulation is chiefly exerted upon the mesentery. Gases and hemorrhagic effusion accumulate in the constricted coil, rendering the twist permanent, and inviting infection, which morbific agents are efficient factors in the subsequent pathological evolution of ulceration, gangrene, and peritonitis.

SYMPTOMS.—Volvulus is frequently met with in individuals of low vitality leading an inactive life, subjects of habitual constipation and flatulent colic. The essential and conspicuous symptoms of volvulus of the sigmoid flexure, the classical seat of this form of obstruction, are pain, constipation, and meteorism. There is so much uncertainty as to the time of the occurrence of the vomiting and collapse that we must relegate these as diagnostic symptoms to a secondary position.

Mode of Onset: It is more or less acute, with marked pain and absolute constipation. The further progress of the case may be confusing in its mildness.

Pain: Complete occlusion does not occur immediately after the rotation of the gut upon its mesenteric axis, consequently there is exaggerated peristaltic action. The initial pain is, therefore, generally paroxysmal in character, expressive of the violent effort to overcome the obstruction. When strangulation becomes absolute the pain is constant, localized, and of moderate severity.

Constipation: Flatus may pass, and slight bowel-movement may occur immediately after the onset of the symptoms. Following this, however, there is absolute constipation; there may be desire to stool, even tenesmus.

Vomiting is an inconstant symptom. It is not pronounced as an early symptom, may not appear until late in the case, or not at all; it is rarely feculent, and but few authorities agree with Ewald, that it is violent.

Shock or **Collapse** varies much in individual cases; the previous state of the health of the patient, the seat of the obstruction, the severity of the pain, and the completeness of the strangulation all influence the degree of shock that attends volvulus. From the usual location of the obstruction at the sigmoid, its remoteness from the abdominal nerve-centers, the immediate impression is not so profound. And as we hold sepsis to be an important factor in the causation of shock, toxines are not elaborated so rapidly or absorbed so quickly from the sigmoid as they are in strangu-

lation of the small intestine. In the onset the pulse is somewhat increased in frequency, the temperature normal, or most probably subnormal. In all cases there is general prostration, but rarely collapse, so characteristic of strangulation of the small intestine. Very soon, however, the symptoms assume the gravest aspect. Pulse, increasing in frequency, is weak and running; respiration is embarrassed by the rapidly developing meteorism; the anxiety of the patient is distressing; if vomiting occurs it does not afford that relief remarked in high obstruction. Vesical and rectal tenesmus add to the discomfort. The urine is diminished in quantity, but is free from indican.

PHYSICAL SIGNS.—**Meteorism**: Paralytic meteorism appears conspicuously in the last stages of all forms of intestinal obstruction. In volvulus, especially of the sigmoid, *meteorism occurs early;* the location of the obstruction low down in the alimentary canal permits of great accumulation, not relieved by vomiting or the belching of gas. It is not alone the early appearance of the meteorism that is significant of volvulus, but its primary location and its progressive and enormous development. The strangulated coil of intestine is the first to be distended; in a few hours after the onset of symptoms marked tympany may be detected below the umbilicus to the right or left side, and this advances more or less rapidly upward. The dimensions attained are equalled in no other condition except colonic or rectal obstruction. Cardiac and pulmonary embarrassment consequent upon the meteorism are distressing symptoms.

DIFFERENTIAL DIAGNOSIS.—Volvulus occurring in the small intestine cannot be differentiated from other forms of intestinal strangulation.

Sigmoidal twists are not attended, as a rule, by the acute symptoms of strangulation. Its frequent occurrence in individuals of advanced age, the rectal tenesmus, and small amount of fluid retained from enema, and chiefly, the early and advancing meteorism, localize the seat of obstruction. The onset is too sudden, and the symptoms too acute for malignant stricture.

PROGNOSIS.—Upon a mechanical basis and a knowledge of the progressive pathological process we cannot hope for a spontaneous untwisting of the volvulus. All cases must necessarily end

fatally, without operation, and, as Mr. Treves has shown, death usually occurs within six days; one patient lived twenty days.

TREATMENT.—In operating for volvulus it is exceptionally possible to relieve the condition by simply untwisting the coil or coils involved. This is more apt to be the case when the twisted bowel is small intestine. When the sigmoid is twisted it must first of all be delivered through the parietal wound, incised and emptied. If it can then be untwisted, and is in a healthy condition, we can either close the opening and replace the bowel or establish a temporary artificial anus by fixing the sigmoid opening in the parietal wound. The nearness of the obstruction to the rectum makes the latter procedure less essential to prevent the absorption of septic material than would be the case if the obstruction was higher up. It has the advantage, however, of preventing recurrence of the volvulus.

When the twist cannot be relieved or when the involved bowel is sufficiently damaged resection becomes necessary, to be followed by anastomosis or the establishment of an artificial anus, as best meets the demand of the individual case. With an unrelieved twist and sound bowel, anastomosis between the bowel above and that below the twist, with drainage of the twisted coil itself, might possibly meet the indications best in some instances. When resection is not necessary fixation of the untwisted coil or shortening of its mesentery should probably be done to provide against recurrence.

INTUSSUSCEPTION.

DEFINITION.—The passage of one segment of intestine into another immediately continuous.

SYNONYM.—Invagination.

FREQUENCY.—Treves asserts that of all forms of intestinal obstruction at least three-eighths of them are intussusception. Since this statement, surgeons have profited by his excellent teaching to such extent that the diagnosis is more accurately and oftener made than formerly, and later authorities give it a relative frequency of three-fourths of all cases of acute intestinal obstruction.

ETIOLOGY.—The length of the mesentery materially influences the degree or extent of invagination, and, as a careful investigation by Mr. Power, extending through a series of 44 cases, shows its proportion to the length of the body is relatively much greater

in infants. This increased length allows increased range of movement to the intestines and is, perhaps, a predisposing cause. If 50 per cent. of invaginations occur at the ileo-cecal juncture, it is reasonable to infer that the angle of union and the relative diameters of the ileum and colon have some bearing as predisposing causes. In isolated cases intestinal polypi may initiate intestinal invagination; so, likewise, the irregularities of the bowel produced by strictures and neoplasms may lead to intussusception.

The important cause, however, of intussusception is unquestionably associated with perverted and irregular muscular action. It was formerly held that a normally contracted bowel was forced by peristaltic action into a paralyzed segment. Lenbuscher, and Nothnagel by their experimental work have shown us that intussusception is essentially a spasmodic condition, and that a paralytic form is secondary to other conditions. The experiments of Langstaff and of Power with various drugs, especially turpeth mineral, show that an enteritis is produced by these irritants, and that the muscular coat of the intestine is thrown into irregular or localized contractions, separated from each other by intervals of relaxed portions of the intestine, conditions eminently favorable for the development of intussusception; and, as Mr. Maylard says, it is not difficult to take a step further in a clinical direction and assume that certain stimulants lead to localized contraction with resulting invagination. Whether these stimuli act from a distance through the nervous system or directly upon the mucous membrane of the involved bowel cannot be ascertained.

While we must admit that many cases of intussusception occur in debilitated individuals, yet Mr. Treves stated that only 8 per cent. of the patients bore evidence of intestinal irritation. The theory of spasm cannot then be applied as a prevailing or exciting cause.

Prolapse of the rectum and ileocecal invagination are alike common in childhood, and in the language of Leichtenstern, "if we consider that the ileocecal opening is distinguished by a sphincter, the contraction of which can be increased to powerful tenesmus, we recognize that there is complete analogy between the condition of invagination in the region of the cecum and the

different kinds of prolapse of the rectum." But a more potent factor than all this, perhaps, in the production of invagination in this region, is the union of the active ileum with the passive colon.

PATHOLOGY.—This very common form of intestinal obstruction may occur at any part of the alimentary canal, and is denotive of the part involved by classification into *enteric, ileocecal, colic,* and *rectal.*

Leichtenstern distinguishes two kinds of intussusception; (1) invagination of the death-struggle, the agonic type, "an unimportant cadaveric incident"; (2) pathological invagination, which is always attended by inflammation and is productive of distinct symptomatic phenomena.

The relative frequency of the various forms shows that ileocecal greatly predominates. It occurs in 44 per cent. of intussusception for all ages, and in from 65 per cent. to 75 per cent. in children. The enteric form is next in point of frequency, about 30 per cent. for all ages.

All parts involved in invagination, for convenience of description, and to the confusion of the student, are named. If a vertical section be made through the intussusception, it will be found that between the central lumen of the bowel and its exterior there are three layers of intestine on each side; the first, the encasing layer, is called the sheath or *intussuscipiens;* the enclosed cylinder of the bowel is known as the *intussusceptum,* which is composed of two layers, the innermost, the entering layer, and the outer or returning layer. Between the opposed peritoneal surfaces of the entering and returning layers is engaged the mesentery. The *apex* of the intussusceptum is the distal end of the invaginated part, definitely marked where the entering layer turns to become the returning layer. The *neck* is the constricted part where the returning layer turns to become the sheath.

Mode of Growth: A very small part of the bowel may be primarily invaginated; but the tendency is for the invagination to gradually increase, and in all forms of intussusception, except ileocolic, this growth is at the expense of the intussuscipiens; and as Mr. Treves aptly illustrates, if the jejunum is invaginated into the ileum, if the mass increases in length it will be solely at the expense of the ileum; no more of the jejunum will become involved.

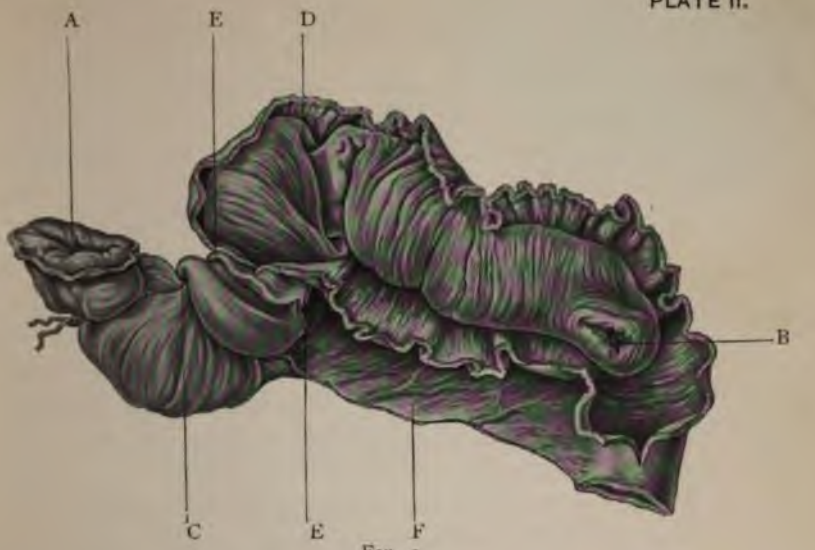

FIG. 1.

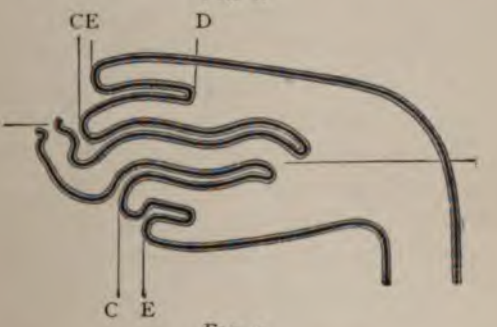

FIG. 2.

ILEOCOLIC INTUSSUSCEPTION.

DOUBLE INVAGINATION.

Fig. 1, Specimen. Fig. 2, Diagram.

The letters have the same significance in both figures.

A. Proximal end of gut; the leader from A extends into the gut lumen now closed by ligature.

B. The apex of the intussusceptum, where the included gut turn upon itself, leaving crescentic pervious aperture; by reference to the accompanying diagram (Fig. 2) it is obvious that the leader from B, if continued, would emerge at A.

C. The neck of the intussuscipiens for the innermost coil, turning back upon itself at D.

D. Ileocecal valve, through which the ileum is invaginated; this is the apex of the middle coil, through which passes the innermost coil.

E. The neck of the outermost coil—the intersussuscipiens. This layer is the caput coli.

The innermost layer enters at C, is covered throughout its entire extent to B, where it turns upon itself, showing a layer from B to D; it passes under the fold of gut at D, to become visible again at C, turning upon itself to enter again at E, showing its deeply congested and veined course to the left of E, and reappearing at D where it turns back to E, again turning upon itself here to become the outermost investing layer which is split open and turned back to show the coils enclosed by it.

In the diagram, in order to show the coats of the intestines which are in contact with each other, the peritoneal coat is stippled, the muscularis is solid black, and the mucous membrane is represented by a thin black line with cross lines at right angles to the continuous lines.

F. Mesocolon.

In the ileocolic variety, in which a portion of the ileum invaginates through the ileocecal valve, the ileum finds no resistance to its entrance into the spacious cecum and colon, and further prolapse of the ileum takes place. The condition is not dissimilar to an internal hernia.

In the pathological process of invagination the mesentery is by no means a passive agent. Leichtenstern attributes to it several important features. The apex of the intussusceptum is always nearest the mesenteric border; the intussusceptum is sharply bent and does not lie in the center of the enveloping cylinder; the cavity is towards the mesenteric attachment. The orifice of the intussusceptum, that is, the unoccluded lumen of the bowel, is not round; it is a slit or fissure. The action of the mesentery thus greatly increases the tendency to occlusion.

The changes which take place at the seat of invagination are due to the partial obstruction of the bowel and to the strangulation of the intussusceptum. The features of obstruction are frequently conspicuously absent. In a number of experiments performed by Senn, in which invagination was artificially produced, no symptoms of obstruction were observed, and when the animals were killed weeks or months after the invagination had been made, the bowel above was not dilated, yet it was hypertrophic.

The danger after invagination has taken place is to the intussusceptum from constriction at the neck. If there is great disproportion between the invaginated part and its sheath, spontaneous reduction may take place; or the intussusception may remain for months without giving rise to symptoms.

Usually, however, there is sufficient constriction of the vessels of the mesentery to cause an effusion between the peritoneal surfaces, agglutinating them together, thus rendering invagination irreducible. This condition of irreducibility may develop in a few hours, but if constriction is not great it may be deferred for weeks. In acute invagination in which constriction is great, the circular constriction at the neck interferes with the return of the venous blood, and immediately the intussusceptum becomes edematous and swollen. In a recent case of ileocecal intussusception the invaginated ileum and appendix were unaltered; the changes were noticeable only in the middle or returning layer, which was enormously thickened and gangrenous. From the

engorged mucous surface of the intussusceptum hemorrhage occurs, and gives conspicuous clinical feature to this form of obstruction. The points of greatest constriction are where the vessels bend upon themselves, that is, at the apex and at the neck; and it is at these points that the ultimate effects of strangulation are first observed.

One of the interesting pathological phenomena of invagination is the gangrene and discharge of the intussusceptum, the result of strangulation of its circulation. In about 40 per cent. of cases, according to Mr. Treves, this spontaneous elimination takes place. It is not so frequently met with in children as in adults, for the reason that in the young the vascular system adapts itself better to pressure, and all the tissues are soft and yielding. Acute cases progressing to gangrene are fatal. Occasionally gangrene occurring in the course of intussusception is limited and chronic, and nature has time to restore the perfect continuity of the bowel at the neck before there is a separation of the intussusceptum. The fact that the gangrenous portion is encased in a comparatively healthy intussuscipiens protects the patient from peritonitis.

The sloughing in more acute cases usually results in death of the patient from peritonitis before separation occurs. Remarkable instances are recorded in which large portions of the bowel are cast off, the most notable of which is one reported by Mr. O'Connor, in which eleven and one-fourth inches, including Meckel's diverticulum, were cast off. Another case, reported by Bullen, and quoted by Maylard, is a remarkable recovery of a man 79 years old after gangrene and discharge of the intussusceptum. One of the most interesting reported cases is the observation of Prof. Peaslee, who exhibited in the New York Academy of Medicine five feet of intestine which had been passed per anum four months before the death of the patient. He also exhibited the intestines removed from the body of this patient after death; the small intestine was only 16 feet long; the large, five feet, ten inches; the invagination had taken place six feet from the duodenum. Unfortunately, ulceration and perforation at the neck of the intussuscipiens may take place at any time, and to this occurrence our fatal results in these cases are largely due.

In chronic cases the sheath becomes thickened and enlarged,

agglutination takes place with the adjacent intestine and omentum, and a large tumor is thus formed. In these cases there is exaggerated peristalsis of the bowel above the obstruction, and if the tumor is watched, it will be seen to vary from time to time with the rigidity of the spasm. Separation occurs from the eleventh to the twenty-first day; it is more likely to occur in enteric invagination. We are bound to admit upon clinical evidence that it is possible for recovery to result from separation of the intussusceptum.

Accumulation of intestinal contents above the seat of obstruction is one of the most important factors which prevents spontaneous disinvagination, and which determines gangrene of the intussusceptum.

Forms of retrograde intussusception are rare. Double descending invaginations are more common. I obtained from a patient of Dr. O. H. Wilson a specimen of double intussusception, combined ileocolic and cecal.

SYMPTOMS.—The clinical picture of intussusception, running an acute course, is strikingly uniform; in no other form of obstruction is the cause so clearly indicated by the symptoms.

Mode of Onset: The onset is usually sudden, especially so in children. We must divest ourselves of the idea that prodromal intestinal irritation or general debility is a necessary precursory condition. In infants intussusception frequently takes place during sleep.

Pain may be regarded as the initial symptom; it is peculiarly intermittent, paroxysmal in character; the violent colic of intussusception surpasses any abdominal pain that children are subject to, unless it is that of acute perforative appendicitis. The suddenly embraced mesenteric nerves, and the violent peristalsis excited, sufficiently explain the immediate severity of the pain, to say nothing of the inflammatory changes which quickly follow. In this as in all incomplete obstructions (Treves), the pain assumes a paroxysmal type. In cases destined to run a subacute or chronic course the intervals of comparative ease may be surprisingly long. In acute cases, however, the paroxysms occur at shorter intervals, until peristalsis succumbs to paralysis, or gangrene supervenes. The sudden cessation of pain, other symptoms persisting, argues gangrene and the release of the constriction at

the neck, with its usual quickly-fatal termination. At first, location of the pain is not diagnostic; with the appearance of a tumor and surrounding peritonitis, pain and tenderness guide us very accurately to the seat of obstruction.

Vomiting appears at first as a reflex symptom and is not pronounced in enteric invagination. Its persistency depends largely upon the completeness of the occlusion and the degree of strangulation. In acute cases, especially in children, the vomiting is uncontrollable. Vomiting is not conspicuous when diarrhea is present.

Constipation: Paradoxical as it may appear, free bowel-actions are the rule in this form of intestinal obstruction, thus emphasizing the important and often overlooked clinical fact, that mere interruption to the passage of feces is but a small part in the pathological manifestation of intestinal strangulation. The violent peristaltic action excited by the invagination precipitates rapid and frequent evacuation of the bowels, and diarrhea is one of the early symptoms. With the evacuation of the contents of the bowels the character of the discharges changes. Hemorrhage occurs from the mucous surface of the engorged intussusceptum, and bloody, mucous, dysenteric stools may be regarded as a constant symptom of invagination of any part of the alimentary canal. In 80 per cent. of cases blood and mucus appear in the stools (Heaton).

In children, especially, the passage of blood following diarrheic discharge should at once suggest intussusception. In the progress of the case, the contraction of the mesentery, twisting and distorting the edematous intussusceptum, may develop very early complete mechanical occlusion; yet the passage of gas and feces usually continues, arrested only by perforation and peritonitis. Tenesmus, instigated by the violent peristalsis and diarrhea, is a distressingly frequent and conspicuous symptom, invariably more marked the nearer the intussusception is to the rectum, and often associated in children with relaxed sphincter ani, through which the bloody stools flow involuntarily.

Shock or **Collapse**: The violent pain, diarrhea, and tenesmus of acute invagination soon very decidedly affect the general condition of the patient. With the acute onset comes very great restlessness. There is much prostration, sometimes profound

collapse, out of which the patient may not revive, death taking place within twenty-four to forty-eight hours. It is rare, however, for a patient to evince that degree of shock observed in other forms of strangulation. In infants the initial shock is severe, and with a piercing cry convulsions may come on, in which the child may die immediately. The general condition of the patient bears a close relation to the degree of strangulation; the more complete the strangulation or obstruction the more pronounced the constitutional symptoms.

Pulse and **Temperature**: The temperature in this, as in all forms of intestinal obstruction, is low, frequently subnormal, and rarely shows more than a degree of elevation; but should the invagination be unrelieved and perforation with peritonitis occur, a rapid rise of temperature to 105° or 106° is noted. But little significance should be attached to the pulse- or temperature-chart.

PHYSICAL SIGNS.—In the very early stage there is usually moderate meteorism, but with the occurrence of the diarrhea and the depletion by hemorrhage of the swelling of the intussusceptum, the abdominal distention subsides and does not reappear until complete obstruction occurs, or until the development of peritonitis.

A symptom of the greatest importance, because of its frequency and peculiar character, is the development of an abdominal tumor, cylindrical or sausage-shaped, and slightly curved. It may appear in two hours after the onset. The tumor is met with in about four-fifths of the cases of ileocecal invagination, and usually occupies the position of the ascending or transverse colon. It is not infrequent for the tumor to change from the left to the right side after injection; it may even disappear temporarily without reduction, and thus deceive us. In 188 cases carefully investigated by Holt, a tumor was found during life in 68 per cent. in children under ten years of age. It is very difficult to outline the tumor in enteric invagination; but in the ileocecal variety, which is so common in children, the presence of the tumor is rarely missed. It becomes prominent and easily outlined during the paroxysms of pain, and we should always avail ourselves of this advantage in examining the abdomen. Before adhesive peritonitis occurs the tumor is movable. With the descent of the

intussusceptum in the ileocolic and ileocecal varieties, the tumor may be felt in the rectum. It is more frequently met with in this locality in children because of the great proportionate length of the mesentery and the mobility of the intestines. To the touch the rectal tumor may closely resemble the os uteri with its central opening, and when it protrudes from the anus, from its deep purple color and globular form it is not infrequently mistaken for a polypus. Manipulation of the abdomen excites peristaltic action and renders the tumor more perceptible.

We must bear in mind that intussusception does not always run the markedly acute course outlined above. In about 18 per cent. of cases the course of intussusception is chronic, and the usual anatomical variety is the ileocecal. A chronic case is due to the fact that the invaginated cylinder becomes permanently attached to the sheath, and the permeability of the alimentary canal is completely restored after the primary swelling disappears. When the invagination assumes a chronic type the diarrhea subsides or becomes intermittent; paroxysmal pains occur at long intervals, accompanied usually by attacks of disordered digestion; the bloody discharge from the bowels ceases. There is usually emaciation and loss of flesh, and through the thin abdominal walls the peristaltic action of the hypertrophied intestine may be observed. The course of chronic obstruction may be prolonged through many months. In cases of chronic intussusception, severe constipation alternates with diarrhea. These cases may terminate in an acute attack of strangulation with gangrene and separation, and ultimate recovery, or fatally by perforation. Leichtenstern admits the probability of complete recovery without separation of the intussusceptum.

Differential Diagnosis.—In our diagnosis we should always take into consideration the age of the patient, the sudden onset of symptoms, the violent paroxysmal pain, the great restlessness, the diarrhea followed by bloody stools, tenesmus, and the appearance of an abdominal tumor, or occasionally the descending apex felt by rectum. Careful consideration of these points would scarcely permit an error in diagnosis.

The sequence of sudden onset, paroxysmal pain, early appearance of an abdominal tumor, and bloody stools, is not produced in any other intra-abdominal condition.

In **appendicitis** the tumor is not so quickly formed, muscular rigidity is constant, bloody stools are absent, and temperature plays a more important part.

Gastro-Enteritis and **Colitis**: In the onset intussusception is mistaken for the simple bowel-disturbances of children, especially gastro-enteritis and colitis. The sudden onset during sleep especially, the distinctly intermittent character of the pain, in the ultra-acute cases the profound collapse, and if the patient survives the acute onset, the development of an abdominal tumor, for the detection of which it may be necessary to employ an anesthetic, determine the diagnosis.

PROGNOSIS.—The course of the disease in acute cases may be exceedingly rapid, death occurring from shock, or in from five to ten days from perforation, gangrene, or peritonitis. The more chronic cases with varying symptoms last several weeks, or a month or two. Death is finally attributable either to absolute obstruction or to exhaustion. The general mortality is from 63 per cent. to 70 per cent. In infants the mortality approximates 80 per cent. Pilz's and Holt's combined statistics of 362 cases give a mortality of 63½ per cent. The possibility of a spontaneous reduction in the early days, with symptoms of moderate severity, is to be considered; and the most desperate cases may finally recover by gangrenous separation and discharge of the intussusceptum.

TREATMENT.—When due consideration has been given to the use of enemata and of rectal insufflation of air or gas in the treatment of acute intussusception, and when all possible credit has been accorded to the method for the undoubted cures it has affected, the fact remains that it is a blind, unreliable and dangerous procedure. Even in apparently successful cases the certainty of complete reduction cannot be established. Cases relieved by this method could be disinvaginated after abdominal section with as little shock, with greater accuracy and certainty, and with provision against peritoneal infection from damaged areas in the disinvaginated bowel, and with provision against recurrence.

At operation after the abdomen is opened disinvagination is attempted, if the involved bowel is sufficiently sound to justify the procedure. It is useless and exceedingly dangerous to disinvag-

inate a partially gangrenous intussusceptum. In disinvaginating pulling upon the neck of the invagination is contraindicated. If reduction can be accomplished at all it will be by pushing the apex back up the intussuscipiens, much after the fashion that the glans penis is pushed through the prepuce in reducing a paraphimosis. Extreme care and gentleness must be used. Force not only may result in splitting the peritoneal coat of the intussuscipiens, but it may even cause rupture through its wall. If disinvagination is affected recurrence may be prevented by fixing the bowel to the parietes or by taking a reef in the mesentery.

When reduction is not possible or is contraindicated on account of the condition of the intussusceptum, Barker's operation should be performed. The intussusceptum is removed through an incision in the intussuscipiens, on its convex border near the neck, the procedure resembling very closely Maunssell's operation for intestinal anastomosis. If sutures have not been passed fixing the neck of the intussusception great care must be exercised in cutting through the intussusceptum to prevent the inner layer when it is severed from slipping out, and discharging the contents of the upper bowel over the field of operation.

When the intussuscipiens itself is partially or wholly gangrenous it must be excised and with it, of course, the intussusceptum. Following the resection anastomosis may be made or an artificial anus established according to the demands of the case and the judgment of the individual operator.

Obstruction by Foreign Bodies.

Gall-stones producing partial or complete intestinal obstruction represent very fairly the phenomena of intestinal occlusion by foreign bodies. The transit of a gall-stone through the alimentary canal is usually accomplished without incident; in exceptional instances it is arrested in its passage, and either from its presence, or circumstantial pathological effect, intestinal occlusion follows.

Etiology.—Mayo Robson's investigations show very clearly that all gall-stones, while yet in the biliary channels or gall-bladder, may excite peritonitis resulting in paralytic obstruction; or the calculus may be discharged from the body through the natural passages or through some circuitous route by ulceration; the

resulting peritonitis, adhesions, and contractions may cause obstruction at some remote date. But it is the mechanical obstruction due to the arrest of the calculus that now concerns us. It is unusual for a calculus sufficiently large to cause intestinal obstruction to reach the intestines by the ordinary channels; and as the calculi do not increase in size by accretion of salts after entering the intestines, they must reach this canal through a fistulous opening established by ulceration between the gall-bladder and an adjacent coil of intestine. Multiple calculi, by clumping, may occlude the lumen of the intestine; the largest reported number is 15 (Lobstein).

Age: Gall-stone obstruction afflicts persons of advanced age; few instances are reported in which the patient is under forty years old.

Sex: It occurs much more frequently in women than in men, in the proportion of four of the former to one of the latter.

PATHOLOGY.—The stones may lodge in any part of the small intestine; the usual sites of imprisonment are the duodenum, jejunum, or lower ileum two or three inches above the valve. Wherever arrested, the probabilities are in fully 40 per cent. of cases that even after the development of the most severe symptoms, the calculus will be dislodged and the patient spontaneously relieved.

The incarcerated calculus, partially or completely occluding the lumen of the gut; blocks the passage of feces, excites violent peristalsis, impedes the circulation, mechanically irritates the gut, establishes an infective *atrium*, and ulceration with perforation is the ultimate disastrous result. Should the patient survive the immediate effects of occlusion, the extraordinary accommodation of nature is well shown in the behavior of the intestine at the seat of occlusion (Leichtenstern). When the obstruction is not complete the stone may push out one side of the intestinal wall, and form for itself a pocket or diverticulum where it may snugly rest for quite a while. Surely this must be a very unusual incident. Under like circumstances of partial obstruction a stone may be discharged by intestinal fistula through the abdominal wall, such a termination being exceedingly rare.

SYMPTOMS.—In many cases the condition is initiated without premonitory symptoms in apparently healthy persons. Kirmisson

and Rochard, who have reported several cases, state that symptoms referable to the liver were absent. The symptoms of obstruction may follow immediately upon an attack of biliary colic, or may be delayed months or years.

The acuity of the symptoms depends largely upon the point in the alimentary canal at which the stone is arrested, and upon the completeness of the obstruction. If it is lodged in the duodenum or jejunum, for reasons many times expressed in this consideration of intestinal obstruction, the symptoms will be more marked and the course of the disease more rapid than if the obstruction were low down in the ileum.

The previous history may be negative, yet in most cases there is a history of biliary colic, an existing stomach or intestinal affection, when there suddenly develop the symptoms of obstruction, violent pain, early and copious vomiting of decomposed intestinal contents; if the obstruction is in the lower ileum the vomitus may become feculent.

There is usually complete constipation, but this may not be absolute; slight bowel-action may occur.

Profound prostration attends the violent pain and ceaseless vomiting. The secretion of urine is greatly decreased, and indicanuria appears. Meteorism is not marked, and if present is rather limited to the upper zone of the abdomen. A palpable tumor may appear from an impacted gall-stone, and should have its seat in the right side (Lobstein).

PROGNOSIS.—Spontaneous recovery occurs by the dislodgment of the calculus—in 44 out of 82 cases (Naunyn), and in many after the most acute symptoms of obstruction were present. Should this favorable issue not occur, the ordinary duration of the disease is from four to fourteen days, the patient dying from exhaustion or peritonitis.

TREATMENT.—Eighty-five per cent. of spontaneous recoveries in appendicitis do not justify us in awaiting that desirable event nor does the comparative frequency of spontaneous recovery from acute obstruction due to gall-stone, foreign bodies, etc., justify a Fabian policy in this condition.

Senn has stated that obstruction does not occur from obturation until changes have taken place in the bowel wall opposite the obturating body. This is undoubtedly correct and for this reason,

whenever possible, the foreign body should be shifted into healthy territory higher up before incision is made for its removal. When the incision has to be made through the damaged bowel, enterostomy will frequently have to be done, and exceptionally resection followed by anastomosis or enterostomy will be indicated.

Obstruction by Fecal Impaction.

When the forces are insufficient to express the contents of the intestines forward, producing regular bowel-passage once or twice in twenty-four hours, the patient may be said to suffer from habitual constipation. This atony of the bowel may amount to absolute paralysis, under which circumstances the condition of *ileus paralyticus* is said to exist. In order to produce fecal impaction it is necessary for a rather long segment of the bowel to be inert, and usually before absolute occlusion of the bowel by fecal masses takes place there has existed for a long time obstinate coprostasis. This is regarded as one of the most frequent, if not *the most* frequent, cause of intestinal obstruction.

Etiology.—The causes of habitual constipation are manifold. It may be found in a primary debility of the muscular fibers of the intestines, or the intestinal atony may be secondary to overwork. It is only necessary to mention here that habitual constipation follows upon errors of diet, excessive activity of the skin and kidneys, hereditary tendencies, diminished amount of intestinal and other secretions. It is also well known that constipation exists as a symptom of many affections remote from the digestive system.

It is most frequently met with in corpulent persons of sedentary habits; women are more predisposed to it than men, and it is especially common among lunatics and subjects of hysteria and hypochondria. As a natural sequence of this chronic constipation, we may have such accumulation of fecal matter as to result in absolute obstruction.

Pathology.—The initial pathological condition is that of atony, and this is recognized by the dilated thin bowel above the seat of obstruction, which is quite the opposite of the compensatory hypertrophy found in chronic obstruction due to organic causes. The long residence of the fecal matter produces pressure-necrosis, and so-called stercoral ulcers develop. These

may be associated with limited adhesive peritonitis, or may result in perforation, giving rise to general peritonitis.

SYMPTOMS.—The general symptoms of fecal impaction develop somewhat gradually. The discharges become less frequent until finally absolute constipation exists. The patient now complains of general debility, is frequently melancholy, and suffers from headache and anorexia. There is some resistance on the part of nature before yielding to the absolute obstruction, and consequently we may have paroxysms of pain; these are not severe. Nausea is present from the first; vomiting may not appear until late, and then only occasionally; stercoraceous vomiting is one of the closing scenes. There is usually constipation both of gases and feces, although spurious movements of the bowels may occur. The abdomen is uniformly distended, and the meteorism progressively increases. Tenderness is not marked until the supervention of peritonitis over the seat of obstruction. Respiration is embarrassed and the action of the kidneys much disordered. The tongue becomes dry and the breath exceedingly offensive.

An essential diagnostic feature of this form of intestinal obstruction is the formation of an abdominal tumor, due to the accumulation of fecal matter. This is not found in the small intestine, as this form of obstruction involves usually the colon only. Fecal accumulations may form a very large tumor, which is often mistaken for various abdominal tumors. Usually, however, if we investigate the history and note the physical character of the tumor, its painlessness, its absolute dulness upon percussion, irregular shape, conforming somewhat to the part of the intestine in which it lies, its doughy, compressible character, these features are usually sufficient in themselves to make the diagnosis.

PROGNOSIS.—The prognosis of intestinal impaction is bad, not because of the obstruction itself, but because of the general condition of the patient leading to it.

TREATMENT.—In the treatment of fecal accumulation purgatives are, of course, contraindicated. Efforts should be made to dislodge the mass with enemata of simple water, oil, solutions of ox-gall, or molasses. Peroxide of hydrogen, it is claimed, will dissolve fecal masses. Usually these efforts are successful.

Fecal accumulations do not per se demand surgical interference. The formation of a coprolith may necessitate colotomy for its removal, but this should be classed as a foreign body rather than as a fecal accumulation. Fecal accumulation above stricture, benign or malignant, will at times require colotomy, while the bowel pathology may necessitate resection or the formation of a permanent artificial anus.

Post-Operative Obstruction.

Definition.—Acute intestinal obstruction following surgical opening of the peritoneal cavity, either by abdominal section or through the vagina; it occurs immediately following the operation, or during convalescence.

Frequency.—Mortuary reports and the reports of cases give a very incorrect idea of the frequency of this complication. My individual experience, which I presume is not exceptional, warrants this statement. Deaths from it are often attributed to peritonitis, secondary shock, and ether-narcosis. Werth reports 11 cases of intestinal obstruction in 1,000 abdominal sections; Winternitz records 11 in 837 abdominal sections. It is relatively more frequent after vaginal hysterectomy than after other intraperitoneal operations.

Etiology.—There are practically only two causes of post-operative intestinal obstruction: (1) *mechanical,* (2) *septic.*

1. The chief mechanical cause is the result of adhesive peritonitis, local in character, and not septic in origin; uniting a knuckle of the bowel to the wound, to raw surfaces, or to adjacent abraded peritoneal surfaces. That such a plastic inflammation can exist after the most perfect aseptic technic and in the absence of microörganisms is shown by Werth.

Yet mild infective processes resulting in adhesions and anchorage of the gut are common enough. Drains, either of gauze or tubes, especially the former, by pressing upon the bowel may cause limited paresis, which under the favorable condition leads on to complete obstruction. As Glénard and Albrecht have shown us, after celiotomy the intra-abdominal pressure is lost, and there is a tendency for the small intestine to sink low in the abdomen; and this traction upon the mesentery, especially the bundle of connective tissue that surrounds the mesenteric vessels

as they pass over the duodenum, compresses it and thus mechanically occludes it.

2. Post-operative sepsis, resulting in peritonitis, produces paresis of the muscular wall of the intestine; and the resulting intestinal obstruction is the conspicuous symptom of the general peritonitis.

PATHOLOGY.—In mechanical obstruction the etiological pathology, as above given, is demonstrable. The adherent intestine is kinked; or, being fixed, volvulus may occur. Adhesive bands may have formed, about which strangulation may have developed. The adhesion and fixation of the gut interrupts its physiological behavior, and excites abnormal peristaltic resistance, to which perverted action the subsequent pathological process is due. The involved gut becomes strangulated, the proximal side rapidly distends with fluid and gases. The resistance is especially low on account of the recent mechanical violence to which it has been subjected, and infection rapidly occurs, death resulting from exhaustion and peritonitis. In those cases due to duodenal obstruction from traction on the mesentery, the chief pathological change is found in the stomach. Acute dilatation rapidly takes place; there is enormous effusion into the stomach; all absorption and assimilation is immediately arrested; and the patient succumbs before there is evidence of peritoneal inflammation. The neglect to examine the stomach at autopsy, and the further fact that the intestines are found flaccid and the general peritoneum apparently healthy, account, I think, for the very small importance assigned to this very common form of post-operative mechanical obstruction.

Septic ileus occurs with every variety of acute peritonitis. In acute diffuse septic peritonitis the patient succumbs to toxemia with symptoms of ileus oftentimes before gross changes are noticeable in the peritoneum. The intestines are found distended, the serosa is dull and dry; there is no evidence of effusion in such cases. In other instances the infection is less virulent, effusion takes place; the exudate may be serous, sero-hemorrhagic, fibrinous, or purulent. The intestines throughout are enormously distended and contain a quantity of fluid.

SYMPTOMS.—Mechanical ileus has a delayed invasion, from a few days to several weeks after operation. The onset is sudden;

the pain is sharp and localized; tenderness and meteorism are confined at first to one region; the pulse immediately before the accident may be normal—it increases in frequency; the temperature remains low, and respiration is not disturbed; there is constipation of gases and feces; vomiting may not come on at once, but is conspicuous as the case progresses; the appearance of the patient undergoes a rapid and marked change for the worse.

Constriction of the duodenum from post-operative enteroptosis presents a little different picture from the other forms of mechanical ileus. The symptoms develop usually in eighteen to twenty-four hours; frequent vomiting is the first symptom to indicate that the patient is doing badly; the epigastrium fills out, is rigid, and then becomes tympanitic; the pulse increases in frequency hourly, and loses in strength; the patient becomes restless, anxious, and is exceedingly thirsty; there is marked decrease in the secretion of urine—there may be sudden anuria; respiration is embarrassed by the distended stomach; both surface and central temperature remains low; the patient dies usually of exhaustion by the third day after operation.

If a careful observation of symptoms and physical signs is made from the hour of operation, we should distinguish mechanical from septic ileus.

In peritonitis the invasion is early; pain, restlessness, and anxiety are observed from the first; vomiting is one of the first symptoms; meteorism is general—epigastric distention is first noticed; the pulse is fast, it may be wiry; the temperature is elevated, there may be hyperpyrexia; Cleveland attaches the greatest importance to the disturbance of respiration, which is greatly increased and embarrassed in adynamic obstruction; constipation may not be absolute at first; the patient has looked ill from the onset, and does not evince that sudden change noticed in mechanical ileus.

Prognosis.—In mechanical ileus due to adhesions, etc., the prognosis is exceedingly good; Winternitz had six recoveries in seven cases. The early recognition of the condition determines the prognosis.

In occlusion of the duodenum the prognosis is exceedingly grave.

Ileus of septic origin, in whatever way treated, is usually fatal; however, Van Arsdale records three successful enterostomies for septic ileus.

SURGICAL INDICATIONS.—Proper technic is protective against mechanical ileus. Raw surfaces should be covered. The intestines must be protected against injury or exposure during operation. Drains should be avoided unless absolutely necessary, and when used should be so placed as not to compress the bowel.

Massage, inflation, change of posture, and such like means consume valuable time. The essential treatment of mechanical ileus is to reopen the abdomen, and release the embarrassed intestine. In septic peritonitis, with conspicuous symptoms of obstruction, experience has taught us that secondary operations are so uniformly fatal that surgeons are discouraged in undertaking them; and many resort to the old opium treatment. The success of Van Arsdale, above referred to, is unique; *enterostomy* has been repeatedly done without avail. If it is to be treated surgically, *enterotomy* should be preferred.

CHRONIC MECHANICAL INTESTINAL OBSTRUCTION.

DEFINITION.—A partial, possibly gradually increasing, occlusion of the lumen of the bowel.

ETIOLOGY.—The mechanical causes of chronic obstruction:

(1) Stenosis.
   a. Benign.
   b. Malignant.
(2) Invagination.
(3) Obturation.
   a. Fecal impaction, foreign bodies, etc.
(4) Tumors.
   a. Of the bowel.
   b. Of other viscera.
(5) Massed adhesions.

Invagination is the principal cause of chronic obstruction in infancy and childhood; in middle life we find principally benign stenosis and tumors of the abdominal and pelvic viscera, while as age advances fecal accumulations and malignant stenosis come to play the most important rôle. Other causes are rather exceptional.

PATHOLOGY.—The general and special pathology of the conditions leading to chronic obstruction is considered elsewhere. The pathology special to the obstruction itself is limited to a hypertrophy of the bowel above the obstruction with catarrhal inflammation, and in many instances ulceration of its mucous membrane. We have a true hypertrophy of the muscle due to its constant effort in overcoming the obstruction. The bowel is thick and leathery to the feel, while the outlines of the blood vessels are distinct. When fecal accumulation or foreign bodies of any description are present, pressure may lead to perforative ulceration followed by peritonitis local or generalized. In this way, too, intervisceral and external fistulæ may be formed.

SYMPTOMS.—The onset of chronic obstruction is gradual. Indefinite digestive disturbances probably first attract attention, more or less abdominal uneasiness and discomfort are noticed gradually increasing in severity until there are distinct colicky attacks. These attacks become more and more frequent and painful, while the discomfort between attacks augments to an extent that keeps the patient in constant distress.

The obstruction being incomplete and usually situated in the large intestine, vomiting is rather unusual unless there is some complicating gastric disturbance.

Constipation is the rule, though catarrhal and ulcerative inflammation of the mucous membrane above the obstruction, may cause a spurious diarrhœa. The diarrhœal discharges have a very foul odor and contain mucous and sometimes blood. The presence of more than a stain of blood is unusual except in malignant disease. Rectal tenesmus frequently accompanies a low obstruction.

As a result of impaired appetite and digestion and from constant suffering, the patient with chronic obstruction rapidly loses health and strength. When the obstruction is due to malignant disease there is, of course, deterioration of the patient's general health with the development of cachexia.

PHYSICAL SIGNS.—The physical evidences of chronic obstruction are upon inspection a varying amount of abdominal distension with visible contracting coils of intestine.

On palpation we may detect a tumor due to fecal impaction, intussusception or malignant stricture. A very firm palpable tumor is

at times caused by an hypertrophied coil of intestine in action. These tumors may be visible as well as palpable.

On ausculation we hear rumbling and gurgling, frequently audible at quite a distance and said to be most marked over the site of obstruction.

Constipation, colicky pains and intestinal patterns are sufficient for a diagnosis of chronic obstruction. The digestive disorders, meteorism and deterioration of the patient's general health are common to many abdominal diseases, while the tumor, unless due to hypertrophied bowel, is of more importance in determining the primary pathology than for diagnosis of obstruction.

PROGNOSIS.—The prognosis in chronic obstruction is determined by the primary pathology, being good in fecal impaction and very bad indeed in malignant disease of the bowel.

TREATMENT.—The treatment of chronic obstruction is the treatment of its cause, and the several causative conditions are treated elsewhere in the text.

PARALYTIC OBSTRUCTION.

DEFINITION.—Adynamic ileus is that form of intestinal obstruction primarily due to loss of motor power.

SYNONYMS.—Pseudo-strangulation, adynamic ileus, ileus paralyticus.

FREQUENCY.—Paralytic obstruction of central origin is comparatively rare; but as a symptom, the outcome of acute peritoneal inflammation, it is familiar to every surgeon.

ETIOLOGY.—In adynamic ileus there is always paralysis of a smaller or larger portion of the intestines. Henrot, quoted by Treves, assigns the following causes: (1) direct paralysis of a segment of gut due to changes in its walls; (2) indirect paralysis depending upon reflex nerve-action; (3) paralysis as a feature of disease of the general nervous system. Its origin as we meet with it clinically is usually traumatic; it follows perforation of viscera, appendicitis, and is a common post-operative sequela. The trauma permits the entrance and encourages the activity of microörganisms, and to their toxic effect upon the peripheral nerves of the intestines the paralysis is due.

PATHOLOGY.—In adynamic ileus arising from sepsis the source of infection can be ascertained, and the evidences of peritonitis

are exhibited. It is only in acute peritonitis that we have ileus arising without the existence of a mechanical cause. Small segments of the bowel only may be involved in the paralysis, though the peritonitis is general; but it is usual to find the whole intestinal tract greatly distended, filled with fluid and gases, the walls thinned by the distention, the serosa showing changes according to the duration. A patient may die of toxemia, with the symptoms of obstruction, so soon after infection that the peritoneum will reveal but little evidence of the destructive effect accomplished through it.

Symptoms.—While acute in its course, it is nevertheless a passive obstruction. The symptoms are all due to loss of motor power. The pain is, therefore, not as acute or paroxysmal as in acute strangulation. Vomiting is of the regurgitant character; the vomitus is at first clear, then usually becomes brown, and frequently of a pea-green color; in the later stages it is very offensive and copious, though never stercoraceous. Constipation, so far as the patient's ability to pass gases or feces is concerned, is absolute; but through a long rectal tube gases and fluid feces will escape. The distention may at first be localized, but soon becomes general; the symmetrical, rapidly-developing and extensive meteorism shows the general involvement of the peritoneum. Unless the patient survives longer than twenty-four or thirty-six hours there is no detectable peritoneal fluid; and this, as we have referred to, is an important differential point from true strangulation. One of the earliest and most constant symptoms to the end is the partial or complete anuria. The temperature is often low, but usually in the last twelve hours before death it rises to 105° or 106°. The pulse with the onset of symptoms is fast, hourly increases in frequency and loses in strength; it is of the greatest prognostic value. The respiration becomes shallow, and the appearance of the patient shows deficient oxygenation. The mind is frequently distressingly clear; or a restless delirium, toxic in origin, develops early.

Prognosis.—Death usually occurs within forty-eight hours.

Surgical Indications.—Only one form of paralytic ileus is amenable to surgical treatment, that arising from infectious processes. The indicated procedure is to remove the primary focus of infection, and to allay the consequent peritonitis.

Differential Diagnostic Table.

Strangulation by Bands.	Volvulus of the Sigmoid.	Intussusception.	Paralytic Obstruction.
Age: Common in young adults. I have seen the extremes of 3 and 63 years.	In middle or advanced life.	Infants and young children especially.	
Sex: More frequent in males, four to one.	Males more frequently.	Somewhat more common in males.	
History: History of previous peritoneal inflammation sometimes.	History of constipation.	In 20 per cent. of cases a history of intestinal disturbance.	History of peritoneal inflammation.
Mode of Onset: Sudden and violent.	Milder, but rapidly progressive.	Instantaneous or gradual.	Gradual.
Pain: Violent and agonizing, remittent.	Not so severe, paroxysmal.	Severe, paroxysmal, intermittent.	Continuous from the first, but not so severe as in any of the others.
Location of Pain: Umbilical or epigastric regions.	Hypogastric or iliac regions.	General or in right iliac region.	Diffuse over abdomen.
Tenderness: Not localized.	Hypogastric or iliac regions frequently.	Over the tumor.	More marked and general.
Vomiting: Early, persistent, becomes feculent.	Late.	Inconstant; may occur with paroxysms of pain.	Early, frequent, and regurgitant.
Constipation: Absolute from the first.	Bowel-action may occur with onset of symptoms.	Diarrhea not infrequent at first. Blood and mucous discharges characteristic, with tenesmus.	No ability or desire for bowels to move; gas may pass through a rectal tube.
Meteorism: Not marked until after peritonitis.	At first local, then general and extensive.	Does not appear without gangrene or perforation.	Early and uniform distention.
Tumor: Absent.	Absent.	Present in perhaps 80 per cent. of cases.	Absent.
Shock or *Collapse:* Profound.	Often absent.	May be severe or very transient.	General condition very bad, but no sudden collapse.
Temperature: In onset often subnormal, and remains low until sepsis occurs.	Normal, or very slight rise.	Same.	Variable. Fever usually marked.
Duration: Three to seven days.	Six to fourteen days.	In acute cases a few hours, to ten days.	One to three days.

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RETRO-PERITONEAL HERNIA.

DEFINITION.—For the purpose of this article, this term is restricted to the engagement of some portion of the intestines in the normal peritoneal fossæ or in the foramen Winslowii.

Treitz, in 1857, first directed the attention of the profession to this form of hernia. The occasional reports of cases, some of them very carefully studied, occur in the literature, but the elaborate monographs of Jonnesco, in 1890, and of Moynihan, in 1899, are exhaustive treatises, and to these authorities the author makes due acknowledgment for information herein conveyed.

VARIETIES.—A subdivision of the general subject reveals four anatomical varieties: (1) Hernia into the foramen of Winslow; (2) Duodenal hernia, developing in one of the fossæ in the duodenal region; (3) Pericecal hernia; (4) Intersigmoid hernia.

1. **Hernia into the Foramen of Winslow:** Moynihan has collected eight cases of this variety. He holds that to permit the occurrence of this form of hernia there must be some gross congenital abnormality, either in the form or size of the aperture or in abnormal length of the mesentery. The hernia occurs into the lesser sac of the peritoneum, and the orifice of the sac is the hiatus Winslowii. The hernial sac rarely contains small intestine, more usually the colon, cecum, and omentum.

These herniæ may exist symptomlessly, but usually they give rise to the phenomena of intestinal strangulation, and present a fairly well-marked symptom-group, which is set forth very clearly in a case recorded by Mr. Treves. Following the occurrence of the hernia was cramping pain, felt in the region of the umbilicus, with vomiting, which occurred at irregular intervals and upon taking food, for the first two days. During the entire course of the cases reported, vomiting is a constant though not a distressing symptom. There is great prostration and feeble pulse. The characteristic features of this form of internal hernia are the absolute constipation, occasionally great tenesmus, and the development of an epigastric tumor. The tumor, situated in the epigas-

tric or left hypochondriac region, may be tender upon pressure, and resonant upon deep percussion.

2. **Duodenal hernia** occurs either on the right or on the left side. The latter is far more frequent and is the ordinary type, first described by Treitz. The hernial sac may contain only a few loops, or the entire small intestine may be retroperitoneally enclosed. The duodenal fossæ have their anterior wall formed by a serous layer composed of two laminæ, of which one invaginates to form the fossa; the other is a continuation of the layers of the different mesos which enclose this region.

When the hernia develops on the left side, the orifice of the sac is situated to the right in close proximity to the lumbar vertebræ. As the hernial mass increases in size, the orifice is forced backward. This form of hernia occurs, according to Mr. Moynihan, in the fossa of Landzert, that is, the paraduodenal fossa. The free border of the hernial orifice is formed by a reflection of the external layer upon the internal, in which fold is found the inferior mesenteric vein and colic artery. The hernial sac begins to develop on the left side of the vertebral column, and may attain to the size of an adult's head or larger. Its posterior relations depend upon the size of the hernial mass. It lies upon the left psoas, between the pancreas above and the kidney below, and may evolute either upward or downward.

The right duodenal hernia, according to Moynihan, originates in a fossa situated in the mesentery in the upper part of the mesojejunum, called by him the fossa of Waldeyer. It lies in the concavity of the arch formed by the superior mesenteric artery, which surrounds the free border of the orifice, and from which spring the branches that supply the small intestine; these branches are included in the hernial sac. Its orifice looks to the left, its fundus to the right and downward. Intestine entering this pouch would produce an enlargement toward the right, upward or downward. The anterior wall consists of an external and an internal layer of peritoneum. The posterior wall of the hernia rests upon the vena cava, right psoas muscle, right kidney, and right renal vessels.

Duodenal hernia occurs at all ages, most commonly in middle life. It is more common in males than in females, in the proportion of 3 to 1. The anatomical conditions necessary for its pro-

duction, according to Jonnesco, are a dilatable excavation in the peritoneum, a resisting ring, and a movable intestine pressing against the excavation. The immediate causes of hernia are practically unknown.

Jonnesco divides all cases of internal hernia into 4 clinical classes: (1) those accidentally discovered post-mortem; (2) those that give rise to slight digestive trouble; (3) those producing chronic intestinal obstruction; (4) those attended by symptoms of acute intestinal strangulation.

Aside from the symptoms common to intestinal obstruction, a duodenal hernia may be probably diagnosed by the development of a circumscribed globular tumor, usually in the umbilical region, with marked retraction of the colonic area. The tumor develops to the right or to the left according to the form of the hernia, is definitely palpable, and variably resonant. Contracting coils of intestine over the tumor may be recognized by palpation. Exaggerated peristalsis may be both seen and felt in the more chronic cases. The size of the tumor, when obstruction is incomplete, varies from time to time, the symptoms usually intensifying with the enlargement of the tumor. Superficial abdominal veins may be found enlarged. In the acute cases with strangulation, the tumor presents but little alteration in size, the patient quickly succumbing with symptoms of acute intestinal obstruction.

Moynihan records two cases of recovery after operation for duodenal hernia.

3. **Pericecal hernia** may be divided into two groups: in the first the sac is situated between the cecum and the iliac or lumbar wall; the second is the ileo-appendicular variety, a rare form, of which Moynihan has collected four cases. The disposition and relations of the sac vary with the volume of the hernia. The sac is forced behind the ascending colon between the two layers of the mesocolon, and contains a variable length of ileum.

4. **Intersigmoid Hernia**: The opening of the intersigmoid fossa is situated in the line of attachment of the sigmoid mesocolon, near the inner margin of the psoas magnus and in front of the bifurcation of the common iliac artery. Its concavity is directed downward, and its orifice will easily admit the finger. Moynihan has collected two cases of this variety.

The symptoms and physical signs of pericecal and intersigmoid herniæ, acutely strangulated, are those classical of intestinal strangulation. When the hernial mass is large the physical signs are those of a tumor characteristically tender and tympanitic upon deep percussion.

SURGICAL INDICATIONS.—In the absence of a tumor, when confronted with symptoms of intestinal strangulation, the abdomen should be opened in the median line below the umbilicus. In right or left duodenal hernia with a tumor, and in hernia into the hiatus of Winslow, the incision should be made in the epigastrium from the xiphoid cartilage to the umbilicus. In the pericecal and intersigmoid varieties the incision should be made over the tumor.

In all internal herniæ, especially in those of the duodenal variety, the great vessels which form the arch of the hernial neck may preclude enlarging the orifice sufficiently to reduce the hernia. Adhesions, kinks, and intussusception are other difficulties interfering with the deliverance of the hernial contents.

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DIAPHRAGMATIC HERNIA.

DEFINITION.—The protrusion of an abdominal viscus or viscera through the diaphragm into the thoracic cavity.

FREQUENCY.—There are 433 cases of diaphragmatic hernia recorded.

ETIOLOGY.—**Age**: Diaphragmatic hernia occurs at all periods of life. It not infrequently exists as a congenital deformity; it is more common in those exposed to accidents and muscular violence.

Sex: It is met with in both sexes.

The diaphragm is protected against abnormal intra-abdominal pressure on the right side by the liver and kidney, and on the left by the stomach, spleen, and kidney. The yielding and distensible stomach does not give the same protection as that afforded by solid viscera. The right crus of the diaphragm is larger and stronger than the left. Upon the left portion we find two depressions for stomach and spleen, an anatomical arrangement predisposing to pouching of the diaphragm. Anatomically we would suppose that the weakest portion of the diaphragm was along the course of the large vessels, and that this would be the site of hernia; yet congenital defects, imperfections in development in the costal fibers, predispose to the development of hernia through this portion. There is a small triangular space just posterior to the ensiform cartilage on each side, left by the separation of the sternal fibers; these spaces give passage to the superior epigastric vessels, and are lined by the pleura above and abdominal peritoneum below, separated only by a thin layer of areolar tissue, and it is very probable that the hernial sac commences to form in this locality.

Etiologically considered, diaphragmatic hernia may exist as a congenital affection, may be gradually acquired, or may be suddenly developed from traumatism. In those cases in which the hernia exists at birth, the protrusion has taken place into the thoracic cavity either through an opening in the diaphragm from

imperfect development, or else the abdominal viscus pushes the thin and yielding diaphragm before it. Acquired diaphragmatic hernia, in most instances, is probably the outcome of a thin and weak area in the diaphragm; or it may occur through a natural opening, the exciting causes being increased intra-abdominal pressure and muscular violence, such as carrying heavy weights, straining at stool, violent labor, etc. Traumatic hernia may be the result of internal violence causing a rupture or separation in the fibers of the diaphragm, or it may be due to penetrating wounds of the diaphragm.

PATHOLOGY.—From the etiology we find that we have four forms of diaphragmatic hernia: (1) Visceral protrusion through a congenital opening due to imperfect development, usually of the costal portion of the diaphragm; (2) through natural apertures; (3) through wounds in the diaphragm; (4) from pouching or distention of the diaphragm. The last variety has been emphasized by Jaffe, who has collected 12 such cases. In those forms of congenital hernia due to abnormal openings in the diaphragm, there is simply a displacement of the abdominal viscera through these openings into the thorax; there is no true hernial sac. So, likewise, in penetrating wounds or ruptures of the diaphragm that permit the migration of the viscera from the abdominal to the thoracic cavity, the viscera pass through a rent in the pleura and diaphragmatic peritoneum and do not carry the latter membrane before as a proper sac. In other instances in which the hernia occurs through intermuscular clefts or weak areas in the diaphragm, the peritoneum is carried before the extruded viscera and forms a true hernial sac.

Practically, we find two pathological varieties of hernia: in the first and most common variety the viscera lie free in the pleural cavity, and not invested by a peritoneal sac; in the second variety the pleura overlies the peritoneum, which, as a hernial sac, contains the viscera—in such cases the hernia is in the thoracic cavity but not in the pleural cavity. In both instances the neck of the hernia is formed by the constricting fibers of the diaphragm.

Any, or practically all, of the abdominal viscera may herniate through the diaphragm. In order of frequency we find the stomach, large intestine, omentum, and small intestine. The

solid viscera, the liver, spleen, and kidney, may occasionally be extruded into the thorax. The hernia may be spontaneously reducible, or it may become incarcerated. The hernia displaces the heart, compresses the lung, and may become strangulated at the neck from changes in the herniated viscus. Adhesions are not infrequently found between the sac and pleura, or, in the absence of a sac, between the viscera and pleura.

SYMPTOMS.—The diagnosis of diaphragmatic hernia is difficult. Only 7 out of 266 cases reviewed by Lachner were diagnosed during life, yet we hope to show that a careful application of physical signs should overcome the difficulties. The rarity of the condition leads to the error of neglecting its consideration.

The defective formation of the diaphragm predisposing to the production of hernia is, of course, congenital, but we must not confound this with *congenital diaphragmatic hernia.* In the latter condition we may understand that a hernia exists at the time of birth, and the first efforts of respiration are embarrassed by the diaphragmatic deficiency, and by the presence in the pleural cavity of the hernial contents; therefore, these patients usually die immediately or in a very short time after birth with symptoms of respiratory distress. They are cyanosed, the left chest does not expand, the abdominal muscles are brought forcibly into play, and the cardiac apex is displaced to the right; after a few hours' struggle, usually, these patients succumb. In others, they appear not to breathe, though the pulse may be strong; if the cord is compressed the child turns pale. Maychell reports two cases: the first died in three hours after birth; the cardiac displacement was noticeable in both; in one the apex beat was under the right nipple.

In studying the symptoms of many cases of diaphragmatic hernia in adults, we find frequently a history of digestive disturbances, especially great thirst, attacks of vomiting, constipation, and abdominal pain. They also complain of difficult breathing, a sense of oppression and inability to perform manual labor. Following such a history, there develops from some slight predisposing cause more acute symptoms. Patients presenting this history are no doubt sufferers from reducible herniæ; the displacement being only temporary, exciting symptoms of a greater or less degree until the organs return to the abdominal cavity.

Hirsch's patient had digestive disturbances, labored breathing, sharp pains in the left side, and vomiting after eating; these symptoms had existed for years before the occurrence of the acute distress that led to a diagnosis. Flagge's patient had a fall in infancy; he had persistent cough, stooping gait, and frequent attacks of abdominal pain; at the age of eighteen years he died with symptoms of intestinal obstruction; necropsy showed the entire small intestine, caput coli, and appendix in the thorax.

Symptoms in Incarceration: When the viscera become incarcerated in the thoracic cavity, whether it be through a congenital opening or defect, in a diaphragmatic pouch, or through a wound, the symptoms in all are the same, and are both abdominal and thoracic in character. The character of the subjective symptoms depends in a great measure, first, upon the amount of visceral extrusion; secondly, upon the degree of constriction at the diaphragm. If the hernia is large, then thoracic symptoms will be conspicuous. A hernial protrusion with a tight constriction will give rise to symptoms of intestinal obstruction. Usually the patient is suddenly seized with a sharp pain in his left side or abdomen; the pain extends upward to the thorax and cardiac region. He complains of difficult breathing, a sense of suffocation, and may have an attack of syncope. The respiratory movements are short, jerky, and "puffing" (Tyson). The patient is excessively nauseated, and vomits; vomiting is usually a persistent feature. If the stomach is much displaced there is great retching and straining, with but little matter ejected. In one of Holt's cases it was two days before the patient vomited; vomiting then continued to the end, though it did not become stercoraceous. Absolute constipation exists from the first. Shock in the acquired form of hernia may not develop immediately, but as the incarceration grows, vital depression becomes marked. In all traumatic herniæ, and in those in which there is strangulation, shock is immediate and profound. In Fortier's case the pulse was 150 and very feeble, respiration 45, and temperature subnormal.

Physical Signs in Incarceration.—*Inspection:* The patient prefers a sitting or half-reclining position. The respiratory movements are quickened and labored. The countenance betrays

great anxiety. Cyanosis is sometimes marked, indicative of defective oxygenation. There is no expansion of the involved side. In left side hernia the apex beat of the heart is displaced to a varying degree to the right and upward, and there may be asymmetrical distention of the left hypochondrium and left thorax. In some instances the abdomen is found scaphoid and rigid.

Palpation: There is tenderness over the region of the stomach along the costal margin; marked rigidity may be noticed; and the respiratory movement on the left side is conspicuously absent. Dextrocardia is noticed.

Percussion: Percussion will yield high-pitched resonance from the fourth or fifth intercostal space in the axillary line, becoming more tympanitic downward, both front and back. The existence of this large hollow, tympanitic space in the left chest is quite significant. There is no sharp demarcation between lung and tympanitic area. In hernia in the left thorax, there is no cardiac dulness on the left. A fixed area of dulness surrounded by tympany would indicate hernia of a solid viscus, which could further be verified by finding the viscus absent from its normal position. Shifting areas of dulness underlying the tympanitic zone are due to the fluid contents of the viscera.

Auscultation: There is absent or impaired vesicular murmur, depending upon the size of the hernia, yet Hirsch found normal breath-sounds notwithstanding that the entire stomach was in the thorax. Vocal fremitus is impaired over the whole compressed lung. The heart's action is rapid and tumultuous, and is heard in its displaced position. Gurgling and ring-sounds are heard over the involved side, not synchronous with respiration.

Other Diagnostic Tests: In a case of supposed diaphragmatic hernia the diagnosis was established very ingeniously by Hirsch. A rubber tube filled with quicksilver was passed into the stomach; the radiograph showed it folded upon itself, and occupying a tympanitic zone in the left chest. In two instances the displacement of the stomach and constriction of the esophagus would not permit the passage of the stomach-tube. Professor Araglino has rendered the diagnosis of diaphragmatic hernia somewhat simpler by employing Lieberstein's method of insufflation of the intestines. He noticed that the left thorax in-

creased simultaneously with the pumping, and by the introduction of water with an ordinary syringe, dulness was developed in areas which had been clear before.

In hernia into the right thorax the symptoms and physical signs would be practically the same, with this exception, that we have sinistrocardia and the signs of hepatic displacement, if that organ participates in the hernia.

DIFFERENTIAL DIAGNOSIS.—**Intestinal Obstruction:** The conspicuous symptoms of diaphragmatic hernia may be those of intestinal obstruction. Many operations have been performed with this diagnosis, seat of obstruction not found, necropsy showing diaphragmatic hernia; Cordier records such an instance. An examination of the chest, and due attention to the physical signs there elicited, are the only and sufficient means of differentiating the two conditions.

PROGNOSIS.—As we have shown, patients may live for many years and suffer from diaphragmatic hernia without strangulation; but with constriction and impeded circulation, marked symptoms occur, and death usually supervenes in from one to five days.

SURGICAL INDICATIONS.—The treatment is essentially surgical. Galassi and Teneri record probably the first operation for diaphragmatic hernia, in 1885. Surgeons are somewhat divided as to the route of operation; Postempski prefers *thoracotomy*. Von Horoch records 4 cases of diaphragmatic hernia following stab-wounds, treated by thoracotomy. *Laparotomy* is the generally preferred method of procedure. A sufficient number of cases have not been operated upon to justify conclusions concerning the technic.

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MESENTERIC THROMBOSIS.

This infrequent condition is an ascending obturation of a mesenteric vein, probably of mycotic origin. So late as December, 1901, Mr. Maylard remarks that there are no cases of this kind recorded in literature. In 1894, Johst recorded 8 cases collected from the general literature, to which he added one of his own. The matter was the subject of an especially instructive paper by Köster, in which he recorded two cases of his own and added several to Johst's list.

In an analysis of the several cases before us, we find varied but kindred causes assigned: intestinal disease, especially typhoid ulceration, long-continued illness, cirrhosis of the liver, and other conditions which impede the circulation. It is probable that it appears most frequently as a post-operative complication, an accident not necessarily restricted to abdominal operations. Our knowledge of the true origin of mesenteric venous thrombosis is somewhat indefinite. Jakowski, acting upon the suggestion of Pilliet, that its origin was in the diseased processes in the bowel, from which foci microörganisms invaded the intima of the blood-vessels, exciting an ascending thrombophlebitis, experimented in this field upon rabbits and guinea-pigs by injecting micro-organisms into the mesenteric circulation, and in this way produced artificial thrombosis. Why thrombosis followed in some cases of typhoid ulceration and not in others is explained in some measure by the variations in the strength of the circulation in different individuals, and also by the quantity and quality of the toxines produced in the individual case. Duclaux, and Gamaleia hold that a more satisfactory explanation is found in the theory advanced by them, that the nature of these bacterial toxines resembles that of the coagulation-ferments. Eisenlohr records an unusual case in which the autopsy showed thrombosis of the mesenteric vein associated with aortic aneurism. The explanation in this case was the alteration in the mucous membrane of the bowel consequent upon arterial changes.

The superior mesenteric vein is the channel most usually affected. Köster says that there is no reason, anatomical or otherwise, why the lower abdominal veins are not involved.

The changes found as the result of thrombosis are: occlusion of all the veins from a certain portion of the bowel; the coils of intestine involved are distended, discolored, of a wine-red or purple hue; the mesentery, usually a somewhat triangular portion, is traversed by numerous extravasations of blood, and is thick and edematous; the peritoneal coat of the bowel has lost its endothelium; necrotic areas appear upon both the serous and mucous surfaces. The extent of bowel and mesentery involved may be only a few inches; or, as in Johst's case, the entire small intestine may be affected. There is rapid accumulation of serosanguinolent fluid, oftentimes almost pure blood, in the peritoneal cavity.

Mesenteric thrombosis is, as a rule, a very rapid process, pursuing its course to a fatal termination, generally within three days, but instances are recorded of longer duration. The onset is marked by severe pain, usually felt in the lumbar region at first, then about the umbilicus or general to the abdomen. In Picqué and Grégoire's case the pain was terrible, and the patient walked the floor or lay doubled up, screaming with agony, without rest day or night. In others, though the pain was less, we note that there was undue and inexplicable restlessness and excitability. Vomiting usually attends the onset of pain. At first the abdomen is neither tender nor distended. An absence of rigidity even has been observed in the first stages. But very soon the abdomen becomes quite tympanitic, the character of the vomitus changes, and not infrequently it becomes coffee-colored from admixture with blood; and while absolute constipation is the rule, in several cases we find recorded diarrhea, and in some, bloody stools. The patient's general condition rapidly weakens, and he passes into collapse or coma.

Köster regards the diagnosis as hardly possible *intra vitam*. The only case that we find in which an approximately correct diagnosis was made was that in which Eisenlohr located the trouble in the mesenteric artery, supposing it to be embolic from the aortic aneurism; the autopsy showed it to be in the superior mesenteric vein, due to intestinal ulceration.

The symptoms in mesenteric thrombosis closely resemble those of acute ileus, especially intussusception, and of perforation-peritonitis. The diarrhea and bloody stools eliminate peritonitis, but heighten the resemblance to intussusception. A significant and distinguishing feature from this form of ileus is the rapid accumulation of free peritoneal fluid, which we find is one of the striking features of mesenteric thrombosis. In cases in which there are known causes of obstruction to the circulation, if abdominal symptoms develop after operation, we should apprehend the possibility of mesenteric thrombosis.

Though some cases run a surprisingly protracted course, all are certainly fatal, and the condition appears to be beyond remedial measures.

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CARCINOMA OF THE INTESTINES.

DEFINITION.—An atypical epithelial neoplasm of glandular origin, usually of the cylindrical-celled type.

FREQUENCY.—In hospital mortuary statistics cancer of the intestines is found once in about three hundred cases (Bovis).

ETIOLOGY.—Carcinoma of the intestinal tract from pylorus to rectum assumes practically the same type; although it occurs with preponderating frequency in the pylorus and rectum, since the rectum properly belongs to pelvic surgery, it is not considered in the domain of this work. Carcinoma is met with most frequently in the large intestine, rarely in the duodenum, and occasionally in the jejunum and ileum. Ewald's table, compiled from statistics of various authors, shows 874 cases in the rectum, 148 cases in the colon, 64 in the cecum and appendix, 26 in the ileum, 19 in the duodenum, 17 in the jejunum. This shows the relative distribution of intestinal carcinoma. The cecum, sigmoid, and rectum are the most frequent parts of the large gut involved. Cumston and Van der Veer have lately collected from the literature 83 cases of carcinoma of the cecum operated upon, to which they add two of their own. There may be some connection between the arterial supply and the determination of the deposit of carcinoma. The points of election, so to speak, are supplied by terminal vessels, and furthermore, they are dependent portions of the intestine, in which we may have impaction, and stercoral ulcers formed. The analogous etiology of carcinoma of the intestine and carcinoma formed upon gastric ulcer has been emphasized by Nothnagel. Habitual constipation, dysenteric and syphilitic ulceration, and alcoholism, may all play minor parts in the production of cancer.

Age: Carcinoma occurs most frequently between the ages of forty and sixty years. An interesting advance in our knowledge from recent observation is that cancer in general, and especially cancer of the intestines, is occurring in younger subjects than it formerly did. Garrard reports a case of carcinoma of the sig-

moid flexure in a child 12 years of age; this patient is perhaps the youngest on record. Czerny reports one of 13 years.

Sex: It is somewhat more common in men, but this point is of no especial value.

PATHOLOGY.—Carcinoma is usually single, though instances are reported of its simultaneous development in different parts. It is generally primary, and rarely invades the intestines by metastasis, although it may by contiguity of tissue. I have twice observed secondary carcinoma of the small intestine occurring from apposition to a carcinomatous uterus.

Varieties: Histologically all forms of cancer of the intestines may be described as cylindrical-celled carcinomata. Harrison Cripps has very clearly shown that the usual malignant disease of the rectum is of this character; and the rectum is not so entirely unlike the rest of the intestinal tube that neoplasms developing in it should be of different histological character. While we do not deny the existence of the medullary, colloid, and scirrhous forms, yet Mr. Treves was unable to find any specimen of scirrhus in the museums, and says, "I am much disposed to doubt its existence." Butlin says that the columnar-celled type is the only form of primary carcinoma found in the intestines, though the shape of the cells may be modified by various conditions. It is remarkable how frequently carcinomata of the intestines take on colloid changes, especially in the young. The hard, annular carcinoma has the physical appearance of scirrhus, and such diagnosis has been frequently made without microscopical examination. The disease may begin as a nodule, a plaque, or a villous projection, but usually as an annular constriction. The entire thickness of the wall of the bowel becomes infiltrated, and there may be extension to opposed structures, leading to fixation.

Curtis makes a most practical working classification of carcinoma: (1) those carcinomata which form a distinct tumor of variable size; these, he says, are likely to be colloid, but, as we have shown, they were primarily cylindrical-celled, and have taken on this colloid change. They are most frequently found in the neighborhood of the ileocecal valve, and although of large size sometimes, they do not materially obstruct the passage-way. The morbid changes commence in Lieberkühn's glands; the direc-

tion of the growth determines their polypoid or sessile character. The edges are usually well-defined; the surface is irregular and often ulcerated. Such growths usually produce a palpable tumor.

(2) The annular form, in comparison with which all other types are infrequent, encircles the bowel in the course of the blood-vessels. This is the typical malignant stricture of the bowel, and while it is a tumor in the sense that it is a neoplastic growth, it does not produce an enlargement; the diseased part is actually smaller. It encircles the gut by infiltration, and narrows and constricts it. The band, or the involved area, is usually very narrow. The proliferation of epithelium is accompanied by abundant connective-tissue stroma in the submucous coat, contracting the lumen of the gut, constricting the blood-supply of the involved area, and developing a tendency to ulceration and necrosis. The involved intestine viewed from without shows simply a groove or constriction, as if it were tied with a piece of tape or thread. A longitudinal section will show all the coats of the bowel drawn in. The indurated area is easily felt between the fingers at operation or autopsy.

Changes in the Intestine: The tendency of all carcinomata, whether annular or tumorous, is to lead to constriction or narrowing of the lumen of the intestine, retarding the passage of the intestinal contents, which resistance nature endeavors to overcome by compensatory hypertrophy. As the constriction tightens the hypertrophy yields to dilatation, and we have in the intestine above the constriction changes analogous to those which occur in the stomach secondary to pyloric obstruction, with these exceptions: the mesenteric attachment prevents uniform dilatation, and hence we have sacculation or pouching of the intestines; the contents of the intestine are more solid and unyielding, and accumulate in these pouches; the circulation of the intestine is more easily disturbed than that of the pylorus; the lowered resistance of the tissues renders them more susceptible to infection from intestinal contents; stercoral or fecal ulcers not infrequently occur, and at times even gangrene may follow at or above the stricture. Inflammation extends to the serosa, and to it adjacent structures may become adherent, so that even in the annular variety, where there is primarily no tumor, a tumor may

form, composed of agglutinated structures; and only by the careful separation of its component parts do we reach the essential morbid anatomy.

The rigid constriction, the small bowel below, and the dilated bowel above, is a mechanical condition favorable to intussusception. Pendulous tumors by their weight may produce this complication, which is not an infrequent occurrence, giving rise to symptoms of acute obstruction with a palpable tumor. In the progress of the case ulceration and perforation may occur above the stricture before there is complete occlusion of the lumen of the bowel, and in this way the case may terminate by fistula, abscess, peritonitis, or sepsis. The intestine on the distal side of the constriction is flaccid and atrophied, and, as some one has remarked, the difference in the size of the proximal ends after excision renders anastomosis more difficult. The condition of the intestines on the distal and proximal sides of the stricture guides the surgeon to the seat of disease.

Glandular Involvement: Involvement of the lymphatic glands is very slow; in many instances it is reported as absent, yet if careful search is made the mesenteric and retroperitoneal glands will be found involved.

Metastases rarely develop in other portions of the intestine; they occur more frequently in the liver, kidneys, and mesenteric glands. Ascites may result from peritoneal irritation and lymph-stagnation.

SYMPTOMS.— As carcinoma of the intestine is an insidious and variable disease, which may elect as its site any point in the intestinal tract from duodenum to rectum, occurring any time after thirty years of age, occasionally in younger individuals, showing no special preference for sex, and pursuing a course beset with accidents, it must produce symptoms variable and often misleading. Yet at some time in the history it becomes apparent that the function of the intestines is seriously interfered with. Arrested absorption, perverted peristalsis, and incomplete evacuation are inevitable functional disturbances, which appear sooner or later as the result of the stenosis from carcinomatous stricture.

The mode of onset is somewhat peculiar and varies with every individual case. The patient may give a history of flatulence

and constipation, extending over a period of one or two years; in other instances there will be a history of frequent attacks of colic, usually attended by constipation, which is relieved by a spontaneous or induced diarrhea. The general condition of the patient may be one of pronounced cachexia with marked emaciation, or he may show no constitutional evidences of disease whatsoever. The duration of intestinal symptoms does not bear a definite relation to the existence of the disease. Occasionally, patients in excellent health will be suddenly seized with acute obstruction as the first evidence of the cancerous stricture.

The various phases above presented are explained in a great measure by the rapidity with which stenosis develops after the beginning of carcinomatous changes. To explain further, if there is a cancer other than one of the annular form, the lumen of the bowel may remain open; the patient then will probably succumb to cachexia and impaired nutrition. On the other hand, in annular constriction stenosis quickly develops, and we have the symptoms of mechanical obstruction before there are any constitutional evidences of the malignant disease. The severity and character of the symptoms and the time of their appearance are materially influenced by the part of the intestine involved—whether the large, or the small bowel. Cancerous growths in the small intestine, owing to the fluid contents of the bowel, do not produce obstructive symptoms as early as those in the large intestine; but when these symptoms do appear, they assume an acute type. This is in obedience to the general law that the higher the obstruction in the intestinal canal the more acute and pronounced the symptoms.

Admitting many exceptions, yet, in a general way, the cardinal symptoms of intestinal carcinoma are: constipation, or constipation alternating with diarrhea, pain, vomiting, exaggerated peristalsis, cachexia; and to these may be added minor and less frequent symptoms hereafter mentioned.

History and Mode of Onset: The previous history of the patient may reveal at some time in life an attack of dysentery, or an infection with syphilis. The cicatrix from a healed typhoid ulcer rarely takes on malignant degeneration. Such facts in the remote history may have an etiological bearing, and should be considered. The present history is usually that of chronic con-

stipation, for the relief of which the patient has resorted to various laxatives. Occasionally this constipation becomes obstinate, and is relieved only by active medication or a spurious diarrhea. These attacks increase in frequency and severity as the stricture tightens, and are precipitated by errors in diet or the lodgment of some indigestible substance, especially seeds of fruits or of berries. This condition of chronic constipation alternating with diarrhea is one of the most characteristic and common features of intestinal stricture; it indicates a progressively narrowing stricture. When guided by a history of such attacks, we are far advanced upon the road to a diagnosis. Unfortunately, these valuable sign-posts are wanting in many instances. Without premonitory symptoms of bowel disturbance the patient presents all the symptoms of acute intestinal obstruction; this is especially true if the disease is in the small intestine. As previously stated, the absolute occlusion is generally due to the lodgment of some of the bowel-contents. It may be due to a sudden change in the position of the growth in the bowel; or the weight and rigidity of the cancerous portion of the bowel may cause acute flexion or torsion. Intussusception may take place, causing absolute occlusion.

Pain: Pain occupies a position second to history in importance and constancy. It is of distinctly colicky character. Its association with obstinate constipation is almost constant. It is due to violent peristalsis, nature's effort to force the bowel-contents through the narrowed lumen. The pain is in the region of the umbilicus, and not distinctly localized until complications occur. At first there may be merely discomfort, fullness and flatulence, usually occurring some hours after eating, precipitated by some indiscretion in diet, and anticipated by the patient when he finds himself becoming constipated. Gradually the pain becomes more violent. Vomiting often occurs in the severe paroxysms. The pain subsides and reoccurs at irregular intervals, and is not relieved until the bowels are freely evacuated. In stricture of the small intestine, attacks of colicky pain, without constipation or diarrhea, may be present.

Vomiting: The higher in the intestinal tract the obstruction, the earlier and more constant is the symptom vomiting. At first these attacks are very slight, and may occur only in the parox-

ysms of pain; later, they are more frequent, and the patient suffers sometimes for days from nausea and vomiting. This is a symptom of obstruction, not peculiar to cancerous obstruction. Temporary relief comes with free evacuation of the bowels, but finally when the stenosis becomes complete, or when perforation and peritonitis occur, vomiting becomes continuous and regurgitant in character. Feculent vomitus indicates absolute obstruction.

Visible Peristalsis: The value of this sign in chronic intestinal obstruction has been emphasized by John Wyllie, and by Frederick Treves, yet I am quite sure that its worth as a diagnostic sign, both as to the character and seat of obstruction, is not sufficiently appreciated. Exaggerated peristalsis occurs in every form of intestinal obstruction; in acute obstruction it cannot be seen, and its presence is betrayed only by the severe, griping pains. Visible and palpable peristalsis, producing distinct patterns on the abdominal wall, occurs only in conditions of subacute and chronic obstruction, and especially in chronic obstruction of the lower bowel. The thin and emaciated state of the patient renders these patterns visible and palpable. They are due to the hypertrophy of the muscular coat of the bowel above the seat of obstruction. When the obstruction is only slight and is permeable, only one or two loops immediately above the stricture may be involved. When the stenosis is great, and especially when the obstruction is low down in the alimentary canal, in the flexures of the colon, or in the sigmoid, then the whole bowel above may stand out in rigid spasm, forming coils individually visible and palpable. If examination is made during these spasms, an excellent opportunity is afforded for determining the location of the stricture. Furthermore, if we keep in mind the mechanism essential to the production of these patterns, we have in their appearance one of the most reliable signs of chronic obstruction.

Cancerous stricture is by far the most common cause of chronic obstruction, and its location so near the lower end of the large intestine makes the extent of these spasms visible throughout the colon-circuit. Through courtesy of Dr. Witherspoon, I saw a patient with cancer of the cecum, the shape of whose abdomen during the height of these peristaltic spasms resembled a large

bowl, the colon forming the rim, and the umbilicus the depressed center. In another instance of cancerous stricture at the splenic flexure the transverse colon would rise up as a conspicuous bow across the abdomen; in this case the spasm usually occurred about six hours after eating, and the patient called this ridge his "cat's back," since it resembled a cat's back when arched.

Meteorism: When obstruction becomes absolute, or when perforation and peritonitis occur, meteorism is marked and progressive. In chronic obstruction, however, meteorism is not as conspicuous a symptom, owing to the empty state of the alimentary canal.

Tumor: A tumor is found in about 50 per cent. of the cases. Of 15 cases Boas found a tumor in 8. A mass may be felt in many instances, due to the agglutination of neighboring organs. The meteorism and location of the hepatic and splenic flexures sometimes render the detection of a tumor very difficult. A movable tumor may temporarily escape the most careful palpation, and repeated examinations are therefore necessary, first with the bowels empty, then distended with gas or water to render them palpable. A general characteristic of intestinal tumors is their extreme mobility, but there are many exceptions to this. They do not move with respiration unless attached to the liver, nor do they change their position with change of posture of the patient. Their shape and size may vary at different times with the degree of bowel-distention. The absence of a tumor does not exclude cancer, yet its detection is of great value in confirming the diagnosis.

Condition of the Stools: The appearance of blood and pus in the stools is a valuable sign, but an infrequent one. But little stress can be laid upon the shape of the stool. Of more value is tenesmus, which is quite a common symptom in cancer of the sigmoid. In cancer of the sigmoid valuable information may be gained by inspection through a long sigmoidoscope, placing the patient in knee-chest position.

Cachexia is of slow development; but when the symptoms of stenosis become marked, patients have the rapid emaciation and usual symptoms of cachexia.

Fever is usually present in the late stages, due to sloughing and ulceration, and to the copremia. Enlargement of the peri-

pheral glands does not seem to be as common in intestinal carcinoma as in carcinoma elsewhere.

DIFFERENTIAL DIAGNOSIS.—It is of little practical value whether the stricture be simple or cancerous in character, the indications in both instances are for immediate surgical relief; yet it is a fact that cancerous strictures have a longer duration than simple strictures, or rather they manifest symptoms earlier. The signs and symptoms are practically the same in both. Of greater value is it to determine the location of the cancer. Riegel reports a case that deserves the most careful study; the diagnosis of cancer of the upper portion of the jejunum was made, first, upon the mobility of the tumor, a feature not present in duodenal growths; second, he excluded pyloric cancer upon the presence of HCl in the gastric juice; third, the repeated vomiting after the stomach had been washed out was explained by regurgitation of food-remains at a later period from the dilated duodenum; fourth, the constant presence of bile in the vomit.

PROGNOSIS.—The duration of intestinal carcinoma depends largely upon the degree of constriction. Cases have been known to live three years, but as a rule they terminate within six or eight months. A large proportion of patients with intestinal cancer succumb to obstruction; others die from perforation-peritonitis; and a few only die from cachexia or secondary cancerous complications.

SURGICAL INDICATIONS.—The ideal treatment is excision of the cancer with intestinal anastomosis. In some instances in which removal of the mass is impossible, we must determine upon the sequestration of the involved gut and short-circuit anastomosis, or the establishment of an artificial anus, the latter to be selected only in extreme cases, as the operative mortality is about the same in each.

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SARCOMA OF THE INTESTINES.

DEFINITION.—An embryonal connective-tissue neoplasm with an atypical preponderance of cells over the intercellular substance.

FREQUENCY.—Sarcoma of the gastro-intestinal canal is of rare occurrence. The existence of primary sarcoma of the small intestine is scarcely referred to in literature. Nothnagel's figures show that of 274 sarcomata only 12 were primary to the intestines. E. Libman reports five cases that occurred in two years at the Mt. Sinai Hospital, N. Y., four of which came to autopsy.

ETIOLOGY.—The clinical history of sarcoma of the intestines reveals but little of consequence as to the causation of the disease. A few cases are reported attributable to traumatism.

Age: Ablon has collected ten cases of sarcomata in children, showing that the disease occurs in all periods of childhood, and may be even of congenital origin. The oldest patient known to be affected with sarcoma is mentioned in Madelung's statistics, aged 52 years. It seems to be a disease of early adult life, from twenty to forty years.

Sex: It shows somewhat of a predilection for males. Of Van Zwalenberg's 14 cases, 9 were males, 5 females. In Madelung's table of 14 cases only one was a female.

Heredity: Heredity plays practically no part in its production.

Flexner reported two cases of sarcoma, in 1893, in which he found peculiar bodies, probably protozoa, which might have some causal relation to lymphosarcoma. Libman has recently confirmed this observation, and attaches some importance to them etiologically.

PATHOLOGY.—Primary sarcoma of the intestines belongs chiefly to the round-celled form, though we note that Mr. Treves says that the spindle-celled is the most common variety. Lymphosarcoma deserves especial mention, as it presents rather different clinical features. We find only one case of melanosarcoma reported.

Sarcomata originate and spread in the submucosa, later involving the muscular coats and mucous membrane; rarely do they extend to the serous coat. The infiltration of the wall of the bowel destroys its elasticity, when the pressure of the intestinal contents leads to dilatation. This passive dilatation of the involved portion of the bowel is characteristic of sarcoma. Baltzer asserts that there never is contraction or narrowing of the lumen, even though the tumor involves the mucous membrane. This condition of the bowel favors ulceration and perforation, resulting in fecal abscesses or general peritonitis.

In the majority of cases metastatic foci in the viscera have been noticed, in the liver and kidney most frequently. Lymphosarcoma quickly disseminates throughout practically all the abdominal viscera. Mesenteric nodes and other parts of the intestine are generally involved; the liver, kidneys, peritoneum, spleen, pancreas, adrenals, and bladder all have been found implicated. Glandular metastases occur, but limited to certain regions. Ablon, who seems to have investigated this very closely, found general lymphatic involvement in only one instance. Ascites from peritoneal involvement is not uncommon.

Symptoms.—As the pathology of sarcoma differs widely from that of carcinoma, so does the symptomatology show but little analogy.

General Condition of the Patient: The earliest and most striking characteristic is the effect upon the patient's general condition; there is marked emaciation, great weakness, pale gray or fawn-colored skin, loss of appetite, and perhaps slight nausea. These general symptoms of cachexia manifest themselves before there is any local disturbance. The temperature is variable; there is usually an evening rise of one or two degrees. With peritoneal involvement and a temperature of 103° the condition very closely resembles tuberculous peritonitis.

Condition of the Bowels: The bowels are irregular, sometimes constipated and sometimes diarrheal. The loose condition of the bowels is a most misleading diagnostic feature. Hemorrhage occurs more frequently, perhaps, than in carcinoma, but it is not a constant or essential symptom. Hemorrhage with tenesmus is suggestive of intussusception.

Pain: Pain is usually felt in the umbilical or epigastric region. It is not of the paroxysmal or colicky character of carcinomatous stricture.

Vomiting: Vomiting does occur, but is not an early or diagnostic symptom.

Tumor: This is the most distinctive objective symptom. Many patients discover the swelling themselves. The tumor rarely appears, however, before the well-marked general symptoms. The tumor may attain considerable size without giving rise to any special local disturbance. The peculiar characteristics of the tumor itself are that it is not painful on palpation and permits of rather rude manipulation; that it is of hard consistency with soft elastic areas, even fluctuant; that its borders can be easily outlined and its relation to neighboring parts readily ascertained. In lymphosarcoma we are likely to have multiple tumors. They grow very rapidly; Baltzer says that you can almost see them grow from day to day. They may attain unusual size; in several instances they have become as large as an adult's head in a very short time. They retain their free mobility for a long while.

Visible peristalsis is not observed.

Meteorism: This appears only in the last scene as a symptom of obstruction or peritonitis.

As the result of the local pressure from the tumor and of obstruction to the portal circulation, we have ascites, and edema of the legs and genitals. The same causes produce enlargement of the veins of the abdomen.

DIFFERENTIAL DIAGNOSIS.— **Carcinoma:** From carcinoma of the intestines sarcoma may be differentiated by the points given:

Sarcoma.	*Carcinoma.*
History.—Very brief and indefinite.	Obstinate constipation alternating with diarrhea.
Age.—Early or middle life.	Middle and advanced age.
Cachexia.—Early and progressive.	Often a very late symptom.
Bowels.—Slight constipation.	Diarrhea occasional, after long constipation.
Pain.—Slight.	Severe, paroxysmal or colicky.

Peristalsis.—Not appreciable.	Marked peristaltic patterns seen and felt.
Tumor.—Appears early, grows rapidly, attains a large size.	Often absent; when found is small and ill-defined.
Duration.—A few weeks or months.	May be one or two years.

Tuberculous Peritonitis: Between tubercular peritonitis and sarcoma of the intestines the differential diagnosis may be difficult, if not impossible. Ascites appears earlier in tuberculosis; the tumor of tuberculosis is chiefly formed by agglutinated intestines, omentum and mesenteric glands, is variable in shape and size as influenced by the condition of the alimentary canal. Peristaltic patterns are more frequently seen in tuberculosis. Pulmonary tuberculosis is frequently associated. The peritoneal effusion should show the presence of the tubercle-bacilli.

Appendicitis: Sarcoma of the intestines must also be differentiated from appendicitis. Many instances are reported in which operations for appendicitis have revealed the presence of a malignant tumor. This is not an unfortunate mistake, for the condition calling for operation is one of emergency, yet if the general symptomatology of sarcoma is taken into consideration, one should avoid this error.

PROGNOSIS.—**Duration:** Sarcomata in children run a very rapid course, terminating fatally in two months from their apparent beginning (Ablon). The duration in all cases is exceedingly short; it varies from a few weeks to something less than a year. Frequently death may be caused by the complications of intussusception or volvulus. Perforation is infrequent. Usually the patient dies from general exhaustion.

SURGICAL INDICATIONS.—Excision of the growth with *intestinal anastomosis,* if the condition of the patient permits.

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BENIGN TUMORS OF THE INTESTINES.

Benign tumors of the intestines may be either cystic or solid. The solid tumors are adenomata, fibromata, lipomata, and nævoids. The cystic tumors are gaseous, blood- and lymph-cysts. The individual consideration of these various tumors is unnecessary, as it is impossible from their symptoms to differentiate their several varieties. Their location and size determine the character and degree of intestinal disturbance. They all produce symptoms of intestinal irritation and obstruction.

Adenomata, the most common of all innocent tumors, appear to have their origin in the follicles of Lieberkühn, or the closed follicles. Histologically they are similar in structure to the polyadenomata of Broca or the villous polypi of Esmarch. The usual site of these tumors is the rectum, though they may extend throughout the colon. They vary greatly in size, may be no larger than a pea; Esmarch reports one weighing four pounds. They are usually sessile, or may become pedunculated. There may be only one, or several hundred; I have removed more than three hundred from the rectum and sigmoid of one patient. Quénu and Landel report forty cases of this condition.

Their etiology is obscure. A remarkable instance of hereditary influence has come under my observation. Mrs. M. had for many years a rectal trouble. "Little tumors were seen and felt high up in the rectum. She finally died of cancer of the rectum" is the statement of her physician, Dr. W. A. Atchison, of Nashville, Tenn. I saw three of her children: a boy of twelve years, who had multiple polypi of the rectum and sigmoid, dying of exhaustion; and two daughters, aged respectively 22 and 20 years, suffered for two years from difficult evacuation, muco-hemorrhagic stools, and great tenesmus. From the older I removed over three hundred polypi. They were soft, very red, bled freely, and were sessile. She has since remained perfectly well. Her younger sister, presenting very much the same symptoms, had distributed throughout the rectum and sigmoid some

twelve or fifteen separate tumors; the highest was about fourteen inches from the anus. One mass as large as a man's fist, and resembling very much a bunch of grapes, was attached in the rectum about four inches from the anus, and was so pedunculated that it would partially protrude with each evacuation.

While these tumors are usually classed as innocent, the report upon this specimen by the pathologist, Dr. Louis Leroy, shows their transitional character. "The portion from the edges of the growth showed near the surface a rather regular arrangement of tubular glands, similar in structure to those of the normal mucosa. In the lumen of many of the tubules mucous and goblet-cells were in abundance. As the tubules approached the deeper layers, they became more irregular, branching frequently and involving the muscular growth. Fibers may be seen running between the tubules vertical to the surface. The general structure of the tumor is such as may be found in intestinal adenoma. A careful examination of a number of sections, however, reveals in several locations areas in which the epithelial cells had passed the borders of the tunica propria of the tubules, and started to infiltrate the surrounding stroma. These areas would indicate that the tumor had just begun in places to become malignant, and would doubtless in time have developed into a columnar-celled carcinoma."

Bland Sutton reports an interesting case of *lipoma* weighing two ounces, enucleated from the colon just above the cecum.

Faulhaber records the removal of a *myoma,* the size of a fist, from the ileum, which is the largest tumor of the kind recorded in this situation.

J. H. Nicoll reports a case of intussusception due to a *nævoid* tumor of the intestines, which on being excised was found to consist of "large thin-walled spaces filled with coagulated blood," and was typically nævoid.

Wollheim records a most interesting example of a *cyst of the intestine.* The tumor was about the size of a child's head, was associated with symptoms of constipation, vomiting, and sometimes hematemesis. It was fluctuant and thought to be an echinococcus cyst of the liver. While under observation it ruptured. Aspiration showed hemorrhagic fluid. Immediate operation was

undertaken, and the tumor was found to be a cyst in the serous coat of the small intestine, the result of venous hemorrhage.

Gaseous cysts, obviously of microbic origin, are found in the intestines. The first case diagnosed *intra vitam* in man was by Hahn, who restored his patient to health by laparotomy, excision of all the pedunculated cysts and crushing of the others. They give rise to gastro-intestinal disturbances, with much distention of the abdomen and obstinate constipation. There may be present, also, symptoms suggesting gastric ulcer, pain in the epigastrium, vomiting of black or bloody masses; occasionally symptoms of ileus. Examination of the abdomen in Hahn's case showed elastic resistance to pressure at various points, and tympany on percussion over these points, while echinococcus cysts give a dull sound. The stomach may be dilated and displaced by the tumefied intestinal loops. The affection develops slowly and sometimes lasts for several years, death finally ensuing from marasmus or intestinal occlusion.

The surgical indication is the simple excision of the tumor by *enterotomy;* or, if the attachment is such as to preclude this, resection of the bowel with anastomosis should be done.

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PERFORATING TYPHOID ULCER.

DEFINITION.—Inflammation, necrosis, ulceration, and sloughing of the agminated glands of Peyer and solitary lymph-follicles is one of the most constant lesions in the course of typhoid fever. Usually this process is arrested when it reaches the muscular coat of the bowel; when it extends through the muscularis and serosa a fistulous communication is established between the bowel and the peritoneal cavity, constituting an intestinal perforation of typhoidal origin.

FREQUENCY.—The types of typhoid fever, in their pathological and symptomatic features, constantly vary. The character of food, general condition and surroundings of the patient unquestionably influence the development of certain lesions. The frequency of abdominal complications in our soldiers during the recent war with Spain and the Philippines emphasizes this fact. Without statistics or other proof corroborative except the statements of my surgical friends, I believe that perforation does not occur as frequently in typhoid fever in the South as it does in the North. I do not admit the argument of Noble, of Atlanta, that our practitioners fail to recognize it. From the above reasons we can understand why the statistics of the frequency of perforation in typhoid fever show such variance. Osler found 34 out of 685 cases (4.96 per cent.); and at Johns Hopkins 3.3 per cent.; Fitz gives 6.58 per cent.; Murchison, 11.38 per cent.; Cushing in thirty autopsies at Fort McPherson found six perforations (20 per cent.).

ETIOLOGY.—The occurrence of perforation is not chargeable to any special line of treatment. Under the various treatments employed from 1869 to 1899 at the Massachusetts General Hospital, the perforations in the three decades were respectively, 1.1 per cent., 0.3 per cent., 1.6 per cent., a difference so slight as to make it doubtful if treatment had any effect upon modifying the tendency to this grave complication. It is likewise shown that it

occurs with equal frequency whether the diet be milk, broth, or soft solids mixed with liquids.

There seems to be no definite relation between perforation and the severity of the attack. It occurs with equal frequency in mild and severe cases.

Age: It occurs most frequently in individuals between the ages of twenty and thirty years. According to Elsberg, it is relatively as frequent in childhood as in adult life.

Sex: It is more frequent in men, in a proportion almost three to one. Fitz's ratio is 71 to 29.

Time of Occurrence: In all but exceptional cases perforation takes place during the obvious progress of the disease, and most frequently between the third and fourth weeks, but we must bear in mind that it may occur even after convalescence is well established, days or weeks after the subsidence of the fever, and in the absence of all intestinal symptoms.

PATHOLOGY.—In the pathology of perforating typhoid ulcer we must take cognizance, because of its prognostic bearing, of the general degenerative changes which take place in all the organs and tissues of the body, and especially in the blood itself. But here we are directly concerned with the ulcer in its preperforative stage, and the conditions incident to perforation. In the course of typhoid fever ulceration and perforation are coextensive with the distribution of Peyer's patches and the solitary follicles.

Site of Perforation: In more than 80 per cent. the perforation is found in the ileum; after this it occurs in the cecum, the appendix and the sigmoid flexure in the order named; Cushing found the seat of the perforation to be in the appendix in 9.6 per cent.

Number of Perforations: Ulceration is usually multiple, but perforation is single, although multiple perforations are numerously reported.

Size of Perforation: The perforation varies in size from a small imperceptible opening to half the circumference of the bowel.

Shape of Perforation: Perforations of the Peyer's patches leave an oval or a round opening, with its serous edge sharply cut, yet it may be very much thinned by the extension of the necrotic process in the mucous and sub-mucous tissues of the bowel.

Impending perforation is recognized by the grayish nodules surrounded by an area of inflammation, which appear just beneath the peritoneum and feel like buttons. In fixed portions of the intestines, such as the cecum and appendix, the inflamed bowel may acquire adhesions, so that finally when perforation takes place the extravasation is confined by adhesions to a local area. Usually, however, a typhoid perforation does not acquire sufficient adhesions to protect the general peritoneal cavity, and as a result of the perforation and the escape of the intestinal contents, even in some instances in which there is no appreciable opening, peritonitis follows. The character of peritonitis that is found in operations or autopsy depends upon the intensity of the infection and the duration of life after the perforation takes place. The effusion may be fibrinous, serous, hemorrhagic, or purulent. If the patient lives sufficiently long, suppurative peritonitis is the inevitable result. The more virulent the infection, the sooner the patient dies, and the less evidence do we have of changes in the peritoneum.

The bowel-wall is deeply congested, soft and friable, and covered here and there by patches of lymph. The perforation is easily recognized by the escape of gas and feces, and the deep-blue discoloration around it. Sometimes there is perforation of the serosa without complete destruction of the other coats of the bowel, and we see shreds of tissue bridging the ulcer. Usually at the site of perforation there is more or less effort on the part of nature to surround it with adhesions, but, as Fitz, and Cushing have emphasized, in the movable ileum this is unsuccessful. Indeed, Fitz, and Cushing think that the behavior of the perforation in the cecum and appendix is so different from that in the movable bowel that we should classify typhoid perforations according to their site. Manifestly the prognosis in a circumscribed cecal or appendiceal perforation is quite a different thing from one in the ileum.

Very little has been done in bacteriological examination of the effusion to determine the character of the peritonitis. Cushing in Case III of his report gives a bacteriological report by Dr. Harris, in which the streptococcus pyogenes was found in cultures from the effusion. Commenting upon this, he says that undoubtedly peritonitides of this nature are very fatal, and that

the culture in this case gave immediately a bad prognosis to what seemed otherwise a favorable case.

SYMPTOMS.—The progress of the ulceration from the mucosa to the serosa may be very rapid. Perforation has been known to occur within the first week from the onset of symptoms; but late in the disease the progress of ulceration is rather slow, and from the pathological changes constantly observed, there is a localized inflammation of the serosa, resulting in slight adhesive peritonitis before perforation actually occurs. Cushing emphasizes the fact that this adhesive peritonitis is sufficient to cause abdominal pain and tenderness, not severe, but rather constant, several days before the final perforation takes place; it should at least arouse the gravest suspicion. When we associate this with increasing leucocytosis it is all the more diagnostic, yet we are not prepared to say that it warrants operation. Billroth advises this prophylactic operation in the preperforative stage, but owing to the obscurity of diagnosis, the many chances for error, the gravity of the patient's condition, and the high operative mortality, we cannot advise operation upon such slight grounds; but when operation is done and the findings are affirmative, it is certainly ideal surgery.

The diagnosis of perforated typhoid ulcer is beset with many difficulties. It occurs frequently in ambulatory cases, or in cases in which typhoid is not suspected. In other cases the mental condition of the patient, stupor or delirium, deprive us of all subjective symptoms. The presence of tympanites obscures the objective signs. Intestinal hemorrhage, as we shall see later, presents many features in common. Therefore, we cannot set forth any one symptom or group of symptoms conclusively diagnostic. In many instances perforation of a typhoid ulcer takes place in the absence of suggestive symptoms, and upon the other hand most characteristic and pronounced symptoms appear when no perforation is found; hence the only conclusive proof of its occurrence is the detection of the perforation.

The accident of perforation in its complete symptomatology presents the following clinical features: The patient is usually a male between the ages of twenty and thirty years; in the second, third, or fourth week of the fever he complains of sudden abdominal pain, perhaps attended by vomiting and marked symp-

toms of shock; his face is anxious, lips drawn and pinched, respiration thoracic and hurried; his temperature has fallen to normal or below; the pulse grows fast and flagging, the abdominal muscles become rigid, and tenderness is marked, especially in the right side. In this condition of shock he may die, but generally in a few hours there is reaction; the pain is now more burning in character, belly rigidity begins to yield to tympany, and examination of the blood shows leucocytosis. The bowels are constipated, vomiting returns and is constant, hepatic dulness is lost, shifting dulness is noticed in the flanks, the temperature rises, the pulse becomes faster and weaker, patient is restless, his mind may be clear, and he quickly succumbs to general peritonitis.

If these symptoms were all present, and the history was clearly that of existing typhoid fever, Mears would have been warranted in saying, a dozen years ago, that the diagnosis of perforation was quite easy. It is admitted by all that unfortunately the positive diagnosis is extremely difficult, and it is only by analysis of these separate symptoms that we may ascribe to each its due importance.

We shall consider the symptoms as they appear clinically. The most important and guiding point in diagnosis of typhoid perforation is the knowledge that the patient has typhoid fever; this knowledge we usually possess before being confronted with the complications. Its frequent occurrence in the so-called ambulatory typhoid must be remembered. The anamnesis must be searchingly investigated.

Pain: The sudden onset of severe abdominal pain is the most constant and valuable symptom of actual perforation. The pain is stabbing, cutting or tearing in character and is usually referred to the right iliac region, though it is sometimes felt generally over the abdomen. Occasionally it is referred to the urinary bladder or even to the glans penis in the male. It is especially significant if it is induced by some sudden change of posture, or occurs during vomiting, active intestinal peristalsis, or after indiscretions in diet. The pain is due to mechanical injury to the peritoneum with the escape of irritating intestinal contents. This subjective symptom may be obscured by the patient's stupor or delirium. Unfortunately, perforation sometimes takes place so gradually that

this valuable symptom does not appear, or is so mild that it escapes consideration. As Cushing has pointed out, abdominal pain may exist for days before actual perforation occurs, yet it is not that sharp, sudden pain that is so characteristic.

Tenderness: With the onset of pain there usually develops marked tenderness, and this too, at first is localized in the right side. In some instances it appears as a general abdominal hyperesthesia. Often the pain may not be so acute, yet the tenderness will be marked and persistent. Tenderness, as is also the case with muscular rigidity and shock, is not so constantly present in typhoid perforation as in other similar conditions, probably because of the fact that the general sensorium is benumbed by the disease.

Vomiting: With the onset of pain vomiting occurs in at least one third of the cases. It is especially significant if the vomiting has not occurred before in this illness.

Shock and **Collapse:** Following quickly after the above symptoms, we notice that a marked impression is made upon the general condition of the patient. He presents the symptoms that vary from slight, almost imperceptible shock to the most profound collapse. While all observers admit that shock is not necessarily a constant symptom, and, indeed, many cases are reported in which it is entirely absent, we must bear in mind that shock is a complex condition, and rarely are all the symptoms of shock absent. The general appearance of the patient, his drawn, anxious face, cold perspiration, ashy color with pale lips, short, hurried respiration, weak and fast pulse, perhaps a chill with sudden fall in temperature, are symptoms clearly denotive of shock of a severe degree; and the vital forces may be so lowered as to reduce the patient to a condition of profound collapse from which he may not revive. These symptoms are so associated in the mind of the general practitioner that they must all be present and in sufficient severity before he will declare the existence of shock. To appreciate the minor grades of shock, the medical attendant must be familiar with the case prior to its onset. How often is the increase in the rate of the pulse of 15 to 20 beats, or a transient uneasiness and restlessness of the patient disregarded! And yet in these very minor symptoms we have the evidences of shock. In brief, I presume to assert that shock of some degree is practically a constant accompaniment of perforation,

modified by the mental lethargy of the patient, and the size and manner of perforation. Whether shock is due to sepsis, as Taylor asserts, or to the assault upon the peritoneum, Keen's idea, I think that the latter authority well sustains his position with this argument: "Were it due to sepsis, then the initial shock should be slight and should steadily increase with the increasing sepsis; but, as a matter of fact, the initial shock is the greatest and is followed by more or less subsidence of shock."

Muscular Rigidity: Sudden rigidity of the abdominal muscles is by many esteemed one of the most significant of all the symptoms of perforation. To it Deaver attaches the greatest importance. Cushing rather discredits its value as a sign diagnostic of perforation, and attributes the parietal tension to a part of a general process rather than a local attempt to protect a tender peritoneum. Muscular rigidity is an early symptom. It accompanies the initial pain and tenderness; this must be appreciated to accord to it its full symptomatic value. The general relaxation of the patient under profound shock and his blunted sensibilities may preclude the occurrence of rigidity, or the leakage may be so slight as not to excite peritoneal resentment. Under these conditions there may be an absence of rigidity from the first, but this is most exceptional, for from a study of many of the reported cases we find that they were not seen by the surgeons until several hours had elapsed after the occurrence of perforation, when rigidity had yielded to tympany. If rigidity is not present, consider the conditions that may explain its absence; if present, ascribe to it the greatest diagnostic significance, secondary only to pain and shock.

Temperature: Under the influence of shock we find the surface of the extremities and brow cold and clammy. Cushing tells us that we must distinguish this apparent coldness from the true central temperature. A sudden fall in the rectal temperature is, when found, a valuable sign, but it is often wanting. The surface of the body may be cold, and the thermometer indicate no change in the central temperature. In some noteworthy cases the temperature has dropped from 104° or 105° to below normal. Dieulafoy, quoted by Cushing, considers the drop in temperature an almost infallible sign of perforation. He further thinks that where the perforation is appendicular, and the extra-

vasation walled off, there may be a decline of surface temperature while the thermometer shows a rise. In some of the cases cited by Keen, this fall in the temperature was the only symptom. Out of thirty-five cases Weir found a marked fall of temperature in only two. In fact, the initial fall in temperature is usually overlooked if present for a rise in temperature following perforation is much more frequently reported.

Pulse: The change in the pulse is noteworthy. With the occurrence of perforation it becomes frequent and weak. If shock is profound, it becomes thready and almost imperceptible. With reaction it returns to its previous state. Illustrative of this take the case of McCrae: At midnight, pulse was 120; next day at noon it was 76. Operation revealed extensive ulceration with perforation and a streptococcus peritonitis. There may be great discord between the pulse and temperature, and it usually shows the pulse fast out of ratio to the temperature. It must be borne in mind that we may have good pulse and normal temperature in general septic peritonitis, yet after the reactionary slowing of the pulse, with the advent of peritonitis the pulse grows faster and progressively weaker.

An increase in arterial tension with the onset of peritonitis following perforation has been investigated by Crile and is considered by him to be sufficiently early, constant and characteristic to be of diagnostic value.

Tympany: Tympany in the course of typhoid fever is due simply to inhibited peristalsis. Suddenly developing tympany, following other symptoms of perforation, is due to gas in the peritoneal cavity and to peritonitis.

Fluid Effusion: Usually the peritonitis leads to a fluid effusion. The rapidity with which this forms is surprising; even in 12 or 18 hours after perforation, shifting dulness in the flanks will denote free peritoneal fluid. In circumscribed perforations the agglutinated intestines, omentum, fibrinous and fluid effusions may form a distinct tumor.

Effacement of Hepatic Dulness: The disappearance of hepatic dulness, in consequence of escape of intestinal gases, would naturally be expected, and it is given as one of the stereotyped signs of intestinal perforation; but, as Keen has shown, it is rarely reported.

Leucocytosis: Thayer says that there is not only no increase in the proportion of colorless corpuscles during the course of typhoid fever, but, on the contrary, there is a tendency toward diminution in the number at the height of disease. With development of an inflammatory complication, immediately a marked increase in the number of leucocytes is observed. It would appear, then, that Finney has reasonable grounds for his statement that leucocytosis, coupled with an attack of severe continued abdominal pain, with nausea and vomiting, is one of the most reliable signs of perforated typhoid ulcer. In Finney's patient the leucocytes before perforation were 3,000; after perforation 16,400 per c.mm. A regular daily count of leucocytes must be made in order that we may recognize an increase in case of grave complication. Thayer thinks that leucocytosis may be but transitory, and hence overlooked, as " it disappears concomitantly with the great outpouring of the leucocytes into the general cavity and the development of general septic peritonitis."

Exploratory Operation: The above symptoms and signs point to the diagnosis of perforation, but the only positive diagnostic sign is the discovery of the perforation by an exploratory celiotomy.

DIFFERENTIAL DIAGNOSIS.—In the course of typhoid fever localized peritonitis with adhesions may develop, presenting many symptoms in common with perforation; it is practically the preperforative stage of the ulcer. In other instances infection occurs without perforation, giving rise to general peritonitis. After a peritonitis is fully developed, we cannot determine the manner of its occurrence. Following the teaching of Fitz, and Cushing, we have already emphasized the frequency of appendicular inflammation and perforation in the course of typhoid fever. This requires no special differentiation. The recognition of an appendicular or pericecal abscess in the course of typhoid fever demands the same treatment as under other circumstances. But under the subject of appendicitis we shall discuss the differential diagnosis between appendicitis and the early stage of typhoid fever with iliac pain and tenderness.

Hemorrhage is differentiated in most instances by the absence of pain, tenderness and rigidity. A high salt water enema will get an evacuation and reveal the presence of blood. The possible coin-

cidence of hemorrhage and perforation, which is not altogether unusual, should be kept in mind.

Pneumonia and pleurisy will at times give rise to symptoms closely similating perforation, but accurate investigation, if we only have these conditions in mind, will readily establish the diagnosis.

Hemorrhagic exudation into the abdominal muscles (Zenker) must be kept in mind for purpose of differentiation.

Iliac thrombosis may present symptoms impossible of differentiation from perforation (Cushing, and Finney).

Recurring to the oft-quoted statement of Fitz that perforation occurs without symptoms, and that suggestive and characteristic symptoms appear without perforation, a discussion in the Royal Medical and Chirurgical Society, of London, affords an apt illustration. Herringham reported a case in which the gravest symptoms were present, warranting the diagnosis of perforation, yet operation revealed only a few scybala in the colon, which an enema relieved, and the patient promptly recovered. In striking contrast was Brunton's case, in which there were practically no characteristic symptoms, and yet perforation was found. We must, therefore, bear in mind that simple constipation may mislead us in the diagnosis.

It seems clear that in the presence of the intra-abdominal symptoms above emphasized, errors in diagnosis are possible, yet conditions are found which warrant the exploration. The occurrence of a perforation is followed by symptoms expressive of the progressive pathological process; I therefore attach great importance in differentiation to the sequence of the phenomena.

PROGNOSIS.—The mortality of perforation is 95 per cent., according to Murchison. The recorded cases of mistakes in diagnosis by the ablest men throws doubt upon the recovery of even 5 per cent., so that, practically, we are fully warranted in predicting an inevitably fatal termination. The patient may die in the initial shock, or he succumbs in a very short time to peritonitis.

Contrast this with the result of operation. According to Harte and Ashurst, the reported mortality of operation from 1894 to 1898 was 72 per cent. This was a decided improvement upon the work prior to that time. Since 1898 there has not been much change, though the reported mortality is somewhat less than 70 per cent. Many fatal cases, however, are not reported and a 70 per cent.

mortality is to be expected at the present time in adults. In children from six to fifteen years of age, as shown by Elsberg, the prognosis is twice as good as in adults.

SURGICAL INDICATIONS.—Seventeen years ago, in 1884, Leyden answered the question, Shall laparotomy be performed for intestinal perforation in typhoid fever? in the affirmative for Germany. But Leyden was not a surgeon, and it remained for Mikulicz, later in that year, to present an exhaustive study of the subject, and to record a recovery after operation. It is answered for America by Van Hook who, in 1891, records the first successful operation in America. Too much honor cannot be accorded to Prof. J. C. Wilson for his eloquent appeal for surgical intervention; not a surgeon himself, this eminent practitioner's arguments are all the more forcible, and admit of no rebuttal. The contributions of Abbe, of New York, Fitz, of Boston, Finney and Cushing, of Baltimore, Hugh Taylor, of Richmond, and especially the classical treatise on "Surgical Complications and Sequels of Typhoid Fever" by Keen, have placed the operation upon a substantial foundation, and have attracted attention to the work of American surgeons the world over.

Though in rare instances of perforation, occurring into the free cavity and giving rise to symptoms, the infection is limited by adhesions, no one at the present day would advocate awaiting such a remote possibility even in the face of a 70 per cent. mortality from operation. Every case of diagnosed perforation not actually moribund should be submitted to operation, and where perforation is suspected, exploration should be made. As Woolsey puts it, we need nothing more than the characteristic pain, tenderness in a fixed spot and rigidity to indicate immediate operation, and we do not require both tenderness and rigidity to be present before proceeding to exploration. The risk to the patient from a carefully done exploration with negative finding has been proved to be slight.

The time for operation is the earliest possible moment after perforation. Shock is not a contraindication. The position taken by Blake that such shock as is due to peritoneal irritation is relieved by a general anæsthetic, is correct and shock due to sepsis can best be treated by removing the source of infection.

It takes nice discrimination to make a choice between cases that are simply desperate and those that are hopeless. The patient, how-

ever, should never be denied, through surgical cowardice any slight chance that he has in operation.

A general anæsthetic is preferable; ether should be used even for children. Some operators prefer cocaine locally, but it hardly seems rational unless the patient is only semi-conscious.

Most operators prefer an incision through or just within the right linea semilunaris, but a median incision is easier to make and easier to close. Particularly is this true of the peritoneal layer of the wound. A median incision gives just as good access to the lower ileum and better access to every other portion of the bowel except the cæcum and first portion of the ascending colon, and Keen found the sigmoid colon most frequently involved after the ileum, cæcum and appendix. The median incision is best situated for flushing the cavity, which procedure is often necessary on account of fecal matter that has escaped through the perforation. It is also best situated for drainage which is used by most surgeons in these cases. The perforation is usually single and small and can be disposed of with a purse-string or a few Lembert sutures. If the opening is too large for closure, or if the adjacent bowel for any reason is not in a satisfactory condition for leaving in situ, our choice lies between anastomosis and enterostomy or colostomy. For the former purpose resection will be necessary. Escher got such satisfactory results from enterostomy that he recommended it as a routine procedure. In the early weeks of typhoid, with the patient in fair condition, anastomosis is preferable if the operator is experienced in intestinal surgery. Late in the disease, with a weak patient and no long drawn-out course of typhoid to be looked forward to, an artificial anus would probably be best. With an inexperienced operator it is best at all stages as compared to anastomosis.

When the perforation has been sutured, or an anastomosis done and the operator is not sure of the capacity of the gut for holding sutures, the bowel should be anchored in the parietal wound by sutures, or still better, under the wound by a gauze pack held in place with small catgut.

The general cavity should be flushed when actual foreign material has escaped into it.

Drainage is used by most operators in these cases, but is of doubtful value unless a necrotic area has to be drained and sequestrated.

Fowler's position is not only of no special advantage, but it is in all probability actually harmful in these weak patients.

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CHRONIC TUBERCULOSIS OF THE CECUM.

DEFINITION.—A tuberculous infection, chronic in its course, hyperplastic in its pathological evolution, and confined to the cecum, ascending colon and cecal end of the ileum.

FREQUENCY.—Conrath, in 1898, collected 85 cases operated upon for this disease. To this number Crowder adds four. Five of these cases only were by American operators. The subject has received the greatest attention in Germany and France. In American literature there are only two contributions worthy of note, the excellent address of Senn on "Tuberculosis of the Intestinal Canal," and the classical article of Crowder upon "Cecal Tuberculosis."

ETIOLOGY.—Intestinal tuberculosis may be primary or secondary. A primary infection is the result of introduction of the bacillus tuberculosis into the body by food; the usual media of infection are meats and milk. The occurrence of primary intestinal tuberculosis is denied by some authorities, and no doubt it is of great rarity, yet autopsical investigation seems to have established its claim as an independent and primary affection.

Secondary infection of the intestinal tract, on the other hand, is quite common and is caused by swallowing tuberculous sputa. In 1,000 autopsies in adults dying of tuberculosis, Eisenhardt found that 566 had secondary infection of the bowel. Fenwick and Dodwell, in 2,000 autopsies, found practically the same percentage of secondary intestinal infection.

In either form diseased states of the alimentary canal are predisposing factors. Chronic catarrhal conditions and dysenteric ulceration afford atria for infection. It is said that gastritis and the consequent alterations in the gastric juice act to predispose. It is by no means necessary that a lesion should exist for infection to occur. The bacilli, as well as their spores, may penetrate the normal intestinal wall.

Age: Children and young adults are especially prone to secondary intestinal tuberculosis, while primary infection with local-

ization is more frequent between the ages of twenty-five and forty years.

Sex: Crowder's statistics of 84 cases show 40 males and 44 females; so there is no special susceptibility of the one sex over the other.

PATHOLOGY.—Secondary manifestations of tuberculosis of the intestinal canal are usually widely disseminated catarrhal and ulcerative conditions. The behavior of these ulcerations may lead to surgical emergencies, but the pronounced initial pathological process not only obscures the symptoms, but frequently precludes intervention. It is especially with cecal tuberculosis that we are concerned. For anatomic, physiologic, and embryonic reasons unnecessary to mention, the cecum is especially prone to infection. Fenwick and Dodwell found the cecum involved in 85 per cent. of all cases of intestinal tuberculosis, and from this locality it invades the ileum and the colon. Peyer's patches and the solitary follicles furnish suitable soil; the tubercular granulations or nodules springing up in them are the early changes. When cecal tuberculosis develops the question of its localization can only be determined by the health and resistance of the individual. If it occurs during the active progress of pulmonary disease, it will probably pursue a general and destructive course. But if it is secondary to a healed pulmonary lesion, or if it is primary, then the infection is mild and the pathological changes are of the chronic hyperplastic character.

The essential pathological factor is the bacillus tuberculosis. The initial pathological change is in the mucosa, and the ultimate effect and characteristic clinical feature is the development of a tumor. Usually the mucosa is the seat of chronic ulceration; the ulcer is gray in color and is set with tubercles. The mucosa may be only very much thickened and covered with papillomatous masses of varying size. The wall of the bowel is thickened, firm, and irregular in outline from cicatricial contractions. Its lumen may be dilated or contracted; the narrowing usually occurs at the site of the ileocecal valve by cicatricial contraction of healed ulcers. The enlarged and thickened cecum is due to a hyperplasia and not to a compensatory hypertrophy; it is inelastic and rigid from tubercular infiltration. The disease shows a tendency to spread along the

colon and not in the ileum. Fibrous adhesions surround the involved bowel, uniting it to the parietes and to adjacent coils of the intestines, enveloping and concealing the appendix which may be otherwise uninvolved. Should the ulceration prove perforative the preformed adhesions circumscribe the resulting abscess. A fecal fistula usually persists after the evacuation of such an abscess, as the ulceration shows but little tendency to heal. As the disease spreads by the lymphatics and not by the blood, we find first the cecal and later the mesenteric glands involved.

SYMPTOMS.—The patient may appear to be well-nourished and in good health; the majority, however, present the symptoms of disturbed digestion and malnutrition; they are anemic, emaciated, and may have fever. They complain of attacks of pain, colicky in character, occurring at irregular intervals without known cause. The attacks are aggravated if constipation is present. As a rule, diarrhea is more frequent than constipation, although during the course of the disease obstipation may occur, and symptoms of ileus develop. In this, as in all forms of intestinal tumors, diarrhea alternating with constipation is a feature. Melena and muco-purulent stools are noted in some cases. During the attacks of pain, which is usually referred to the iliac region, the patient may vomit distressingly, and at such times the abdominal muscles are rigid. A further examination will reveal a nodular tumor which lies above the brim of the pelvis, usually situated in the ileocecal region. The tumor is hard, frequently movable, usually only toward the middle line; its mobility is one of its diagnostic features. It does not move with respiration, and upon percussion a hollow, impaired tympanitic note is elicited. If observed during an attack of colic the tumor perceptibly enlarges in size and becomes distinct in outline. If the colon is inflated with gas or water the tumor becomes more visible and palpable. Upon auscultation we may hear gurgling or sizzing sounds.

DIFFERENTIAL DIAGNOSIS.—**Appendicitis**: The presence of a tumor in the right iliac region at once suggests appendicitis. The general condition of the patient and the long duration of the symptoms are opposed to this. The persistency of the tumor, its variations in size from time to time, its mobility, its tym-

panitic note, and its response to inflation, with the absence of septic temperature and pulse, should make the diagnosis from appendicitis clear.

Carcinoma of the cecum presents symptoms so identical in nature that it is impossible to differentiate the two, and even after removal the specimen has often been regarded as malignant until subjected to microscopical examination.

PROGNOSIS.—The tendency of this form of tuberculosis is to remain localized for an indefinite time. The complications of suppuration, intestinal obstruction, diarrhea, and hemorrhage are causes of fatal termination. At any time in the progress of the case infection may occur.

Magill records 24 resections of the ileocecal coil for tuberculosis, with only 4 deaths, a mortality of 16.66 per cent.

SURGICAL INDICATIONS.—Chronic tuberculous inflammation of the cecum should in all cases be treated by resection of the cecum with removal of involved glands, provided the patient's strength will permit. The continuity of the intestine is established preferably by suture, or by lateral anastomosis with absorbable bone-plates, or by a terminal or lateral implantation of the ileum into the colon, best effected with the Murphy button.

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APPENDICITIS.

DEFINITION.—An infective inflammation of the appendix vermiformis ceci.

FREQUENCY.—Appendicitis is the most frequent of all intra-abdominal surgical diseases.

ETIOLOGY.—Appendiceal inflammation is essentially dependent upon bacterial invasion for its production, yet there are certain conditions, anatomical, pathological, and mechanical, acting directly or remotely as contributory factors, and these we may regard in the light of predisposing causes, creating a susceptibility, a *locus minoris resistentiæ* open to the assault of micro-organisms.

1. **Predisposing Causes:** It must be borne in mind that the vermiform appendix is a vestigial organ undergoing an evolutionary and a retrograde metamorphosis, and it is recognized that structures of this character are especially liable to inflammation.

The next most important anatomical characteristic is the paucity of the blood-supply. The appendicular artery, a branch of the ileocolic, lies between the folds of the meso-appendix along its free border, and from this single artery the distal four-fifths of the appendix in the male receives its nourishment. The appendiculo-ovarian ligament of Clado is an inconstant anatomical structure, and does not always contain an artery. Interference with the circulation is very likely to occur, as we shall show later, and is one of the chief causes of appendicitis.

The appendix is an organ of variable length, from one to nine inches, and is provided with a mesentery which, as a rule, extends along its full length. The appendix, attached to the caput ceci, may be straight or kinked upon itself; and it occupies variable positions with relation to the cecum. Its usual position, under the inner or outer border of the cecum, renders it susceptible to pressure not only from the contents of the cecum, but from many conditions, physiological or pathological, that increase intra-abdominal pressure. Instances of this are found in the

frequency of the occurrence of appendicitis during pregnancy; Gerster reports three such cases. I have found appendicitis in more than one instance associated with fibroid tumor of the uterus. Oftentimes the appendix rests upon the psoas muscle, and this renders it liable to direct violence. More important than pressure, is the effect of torsion upon the circulation. When the mesentery is long the appendix may lie straight from the caput ceci in any of the positions of the compass. It is frequently found, however, especially when its mesentery is short, curled and contorted upon itself. In one instance I found volvulus of an unusually long appendix obstructing its lumen and strangulating its circulation.

More important than the position, however, is the diameter of the lumen of the appendix, and the size of the ceco-appendiceal communication. Treves states that the lumen is often found obliterated. Other observers, Mr. Lockwood especially, entertain very different views. Occlusion of the lumen of the appendix is pathological. Distal occlusion may be from senile retrogression. Narrowing of the proximal end favors fecal stagnation. Fluid feces may normally circulate in the appendix, and the so-called valve of Gerlach, merely a crescentic fold of mucous membrane, is not competent to prevent this. It is the obstruction to the egress of the contents of the appendix rather than the entrance which excites pathological results.

The structure of the appendix in a general way is similar to that of the large intestine, with its peritoneal envelope, thin circular, and thick longitudinal muscular fibers. There is relatively a thick layer of connective tissue with arterial openings and lymphatic depressions. The mucous coat, lined with cylindrical epithelium, contains tubular glands and closed follicles. The most striking histological feature of the appendix is its richness in lymphoid elements. Watney, Ransohoff, and Bland Sutton have all called attention to the similarity of this structure to the tonsil, and show that both are easily susceptible to inflammation in early life; and like the tonsil, the lymphoid elements of the appendix undergo evolutionary and retrograde metamorphosis.

The lymphatics of the appendix pass into the lymphatic appendicular ganglion, which lies in the angle formed by the appendix and cecum. Clado has demonstrated lymphatic channels in the

PLATE III.

Position of Appendix (*After Fowler*).

appendiculo-ovarian ligament. This arrangement explains the extension of infection.

Viewing the appendix as a whole, we find a firm, unyielding peritoneal encasement, embracing structures composed largely of soft embryonal elements, so that with infection and inflammation of these inner structures we have added compression and tension, thus partially explaining the gangrenous tendency of appendicitis.

Age: The frequency of appendicitis in the adolescent has impressed the general physician that it is confined to that period of life. Admittedly it is more frequent between the ages of ten and thirty years, that period in which the lymphoid structure is most abundant and active. What etiological bearing age may have beyond this is doubtful, but certainly in the young the tissues have less resistance, and there is marked tendency for the inflammation to become perforative or gangrenous.

Sex: Fitz, Fowler, and others give the relative frequency in sex as four males to one female. Price, Edebohls, and others contend that there is no such great disparity in the sexes. The appendix in the female may have a better blood-supply through a branch of the ovarian artery when the ligament of Clado is present, and on this account may enjoy greater resisting power. It appears to me that there are better reasons why it is not quite so frequent in females: appendicitis is often mistaken for ovarian or tubal trouble; females lead, as a rule, a more regular, cautious life, and they are not so exposed to traumatism.

Digestive Disorders: Concerning the condition of the digestive system as a predisposing cause, the contradictory and rather vague statements of authors leave the question undetermined. The bowels may be normal, constipated, or there may be diarrhea. I can only state that my observation has been that immediately preceding an acute attack there is no intestinal disturbance that we can look upon as etiological. The digestive disturbance usually assigned as causal is, in fact, the result of already existing appendicitis.

Much evidence has been deduced, mostly by French writers, to show the influence of diet. It is claimed that a meat diet strongly predisposes to appendicular infection. Vegetarians, on the other hand, are supposed to be practically immune.

Constitutional Conditions: In many instances local causes are wanting and we must look for constitutional agencies. The occurrence of appendicitis in rheumatics, and its frequency during epidemics of influenza, have been remarked upon. That these constitutional maladies, rheumatism for instance, may permanently injure lymphoid tissues and cause obliterating endarteritis is well known, and thus far only can they have any causative influence.

Movable Kidney: Since our attention was called to the association of appendicitis with movable kidney by Beck, and Edebohls, I am convinced that they are correct, and that this deserves consideration as an etiological factor.

Foreign Bodies: Dieulafoy is responsible for the original statement that appendicitis is invariably caused by a calculus, or by some accumulation of fecal or foreign matter. This modern endorsement has firmly fixed an ancient but erroneous idea in the minds of the profession, and it seems difficult to eradicate it. Every abdominal surgeon is cognizant of the fact that semi-solid or inspissated feces is found in the appendix, the organ presenting neither pathological change nor symptoms. It is true that the appendix has not the inherent power of expelling its contents, as compared with the rest of the alimentary canal, yet I believe that Kelynack is correct when he says that there is, as it were, an appendicular passage of feces. The accidents of size, position, pressure, etc., further tend to retard the passage of feces. Under these conditions the retained fecal matter loses its watery elements by absorption, and becomes a hardened mass; and as such, as we shall show later on, it may prove harmful.

True foreign bodies are found in only 7 per cent. to 12 per cent. of cases. In my own work, which has been rather extensive, I have only once found a true foreign body; this was determined by most careful analysis to be a gall-stone. The various seeds, pips, etc., so frequently described in literature as found and assignedly causing the appendicitis, are really fecal masses, or true appendicular calculi, moulded into these familiar shapes by the peristaltic action of the appendix in its effort to expel them. Many bodies found in the appendix are neither fecal masses nor foreign bodies swallowed, but they are really appendicular products. The idea of Talamon, that they were formed in the cecum and dropped into the appendix, has long since been abandoned.

Analysis of these bodies shows that they contain but little feces; they consist chiefly of the insoluble salts precipitated from mucus, showing calcium and magnesium compounds enmassed in mucus. These calculi grow by the deposit of mucus, and hence often appear laminated. A most significant point is that they occur between the ages of twenty and thirty years most frequently, a period in which the lymphoid structure of the appendix is most abundant and physiologically active. It is a well-known fact that microörganisms play an important part in the production of gall-stones; the idea is advanced from analogous reasoning rather than from any positive bacteriological examination, that appendicular calculi are the effects of mild infection rather than the cause of inflammation.

Foreign bodies in the appendix, of whatever nature, excite trouble mechanically in one of several ways: the appendix resents their presence, and an extra effort is made to expel them—this is expressed as appendicular colic; the impaction of the foreign body abrades the mucous membrane and creates an *atrium* for additional infection; by its presence it may obstruct the circulation, giving rise to local or general necrosis. And if the theory is correct that the foreign body is oftentimes the product of preëxisting inflammation, we have already a crippled organ which is susceptible to infection. The frequency of these concretions varies from 40 per cent. to 80 per cent.; Murphy found them in 38 out of 40 cases; they have been quite the rule in my experience. However, that they are not an essential factor in appendicitis is shown by the fact that we have all varieties of the disease occurring in their absence.

Traumatism: It is becoming more generally recognized that traumatism, exerted in various ways, plays an important rôle in the production of appendicitis. Small has recently reported 13 cases which he believed were traumatic in origin. It may follow upon a blow upon or contusion of the abdomen. Heavy burdens pressing forcibly against the abdomen may induce it. Straining, especially with the arms extended above the head, is cited as a cause. Foreign bodies within the appendix, as we have shown, may cause traumatism. Resting as the appendix sometimes does upon the right psoas muscle, Byron Robinson believes that the muscular contraction may injure the appendix, and in proof of

this he cites the frequency of appendicitis in athletic men and boys. That traumatism can cause infection of the normal appendix is altogether improbable. What is much more likely is that traumatism lights up a slumbering focus already present.

All the above conditions enumerated we must regard in the light of predisposing causes; oftentimes they exist individually and collectively without producing appendiceal inflammation. Some other factor, then, is necessary for its production, and that is found in the infective microörganisms which we regard as the essential cause.

2. **Essential Cause:** It is established beyond cavil that the bacillus coli communis normally inhabits the intestinal canal after birth, and that it is not found in other abdominal viscera without an associate lesion of the intestine. It is clearly shown by Welch and others that this lesion is not necessarily gross; any condition which impairs the circulation will permit the passage of these germs through the wall of the intestine. The frequent occurrence of the bacillus coli communis in pure culture in cases of exudative appendicitis has been repeatedly recognized. Bacteriological studies of appendicitis are frequently made. Fowler, Welch, Robb, Clado, Hodenpyl, and Beck were the first to establish clearly the etiological rôle played by microörganisms, and now it is no longer denied that appendicitis is an infective process. Whether it is a simple or a mixed infection is not thoroughly established, but all observations point to the ubiquitous bacterium coli commune as the predominating etiological factor. The streptococcus lanceolatus, the bacillus pyogenes fetidus, and the staphylococcus are found either in pure or mixed culture. Hodenpyl found the bacillus coli communis in 34 out of 35 cases of exudative appendicitis, and in 31 of these it was found unassociated with any other germ. The pathogenic power of the bacillus coli communis is shown by its occurrence in lesions unassociated with any other germ, and by the effects of its inoculation into animals in pure culture. These points have been affirmatively settled. Many authorities believe that appendicitis is a mixed infection; Welch maintains that it is due to the combined influence of the bacterium coli and streptococcus, and that the failure to discover the streptococcus does not necessarily prove its absence.

Specific forms of appendicitis are occasionally met with, attributable to their special microörganisms; thus we have the bacillus typhosus, the bacillus tuberculosis, the actinomyces probably.

A summation of our knowledge as to the etiological factors of appendicitis leads us to think that there are predisposing causes, which create in the appendix a condition of susceptibility to inflammation, especially favorable because of the histological structure of that organ. The one needed fact for the establishment of this inflammation, from its mildest to its most destructive type, is supplied in the essential cause, the infecting germ.

PATHOLOGY.—When infection of the appendix occurs a pathological process is initiated which varies with the virulence of the infecting microörganism, the combative power of the phagocytes, the resistance of the patient, and the accidents of the several predisposing causes. The pathological evolution of appendicitis is one of degree and stages merely, and not of kind.

Varieties: I. Non-suppurative Appendicitis:
- (1) Endo-appendicitis.
- (2) Interstitial appendicitis.
  - (a) Hyperplastic.
  - (b) Obliterative.

II. Suppurative Appendicitis:
- (1) Intratubular.
- (2) Intramural.
- (3) Peri-appendicular.

III. Perforative Appendicitis.

IV. Gangrenous Appendicitis.

I. **Non-suppurative Appendicitis:** The types of non-suppurative appendicitis below presented necessarily imply predisposing conditions of moderate influence, and infection of mild activity. This constitutes what is regarded by some as the class suitable for medical treatment; but, as we hope to show, all these varieties create a susceptible pathological state predisposing to recurrent symptoms, and are essentially dangerous in their ultimate effects.

1. **Endo-Appendicitis:** This term is chosen as preferable to *catarrhal appendicitis,* which suggests an inflammation limited to the mucous surface alone; in endo-appendicitis there is an in-

flammation of the mucous membrane and the lymphoid structures—the soft, distensible inner tube of the appendix. To one at all familiar with the histology of the appendix, it is inconceivable that the epithelial lining alone can be involved. In response to the infecting microörganism there is excited a hyperemia and a hypersecretion, as is manifest by the angry red color and the abundant secretion of mucus. The infection may be confined to a small area, or it may be general throughout the appendicular lining. There is swelling and engorgement of the soft elements involved, creating tension and compression by the ensheathing outer wall. To the unaided eye the changes appear insignificant. Microscopically the exudate into the lumen is shown to consist of mucus, epithelium, and crystalline bodies, all filled with bacteria. The submucous vessels are engorged and the submucosa is infiltrated with serum and leucocytes. The effect of this inflammation is to create spasmodic contraction of the muscular coat, as symptomatically expressed in colic. Furthermore, there is occlusion of the lumen of the tube, with retention of the now greatly increased secretion. If the infecting agent is mild, the phagocytosis is effective; if the physical conditions permit the appendix to disgorge its contents, then the process may be arrested here, and the inflammation will subside.

The results of such an attack are grossly shown as strictures in the appendix at the points of flexion. There may be one or more of these—the usual site is near the base or proximal end. These strictures simply show that there has been a superficial ulceration, which, healing, has undergone cicatricial contraction.

Narrowing of the lumen is a fairly constant sequel of endo-appendicitis, the effect of which is to favor retention of mucus and feces, and ultimately to lead to the formation of concretions. Microscopical inspection of a specimen of endo-appendicitis should convince even the most skeptical that an appendix so involved cannot but remain an injured structure, susceptible to renewed infection upon the slightest provocation, or else continue to suffer from the ill effects of chronic or latent inflammation. This statement does not imply that one or two attacks of endo-appendicitis may not entirely subside, and the patient remain symptomatically well ever afterwards. It is rather my purpose to show that the pathological effects caused by a mild

attack create new predisposing causes, and render the organ especially vulnerable.

If the inhibiting conditions above mentioned are not sufficient to overcome the infection, the germs penetrate the submucosa through the channels of the lymphatics, and now, as Beck says, the breach being shot, there is no further halt to the progress of the infection until structures of greater resistance are encountered.

2. **Interstitial Appendicitis:** This is by some denominated parietal appendicitis. This variety is restricted to appendiceal inflammation of a non-suppurative type which involves the entire thickness of the wall of the appendix. This inflammation, chronic in its course, hyperplastic or obliterative in its pathological effect, and remittent in its symptomatic expression, necessarily implies mild infection with marked resistance. The infecting *atrium* in the mucosa permits the entrance of microörganisms. They perhaps rapidly invade the mucosa and lymphoid structures, giving rise to symptoms of acute endo-appendicitis. The acuteness of the attack subsides, but the infection, now less virulent, continues. The microörganisms, traveling by the lymphatics especially through the mucosa, permeate the open spaces of the muscularis, reach the serosa, and may be arrested here; but usually a reinforcement of leucocytes, which the surrounding peritoneum abundantly supplies, is required to limit the progress of the infection.

There are two distinct forms of interstitial appendicitis: (a) hyperplastic, (b) obliterative.

(a) *Hyperplastic:* In the hyperplastic form the organ appears enlarged and thickened, and the meso-appendix participates in this general hypertrophy. The lumen of the appendix is usually dilated, either uniformly or cystically, between points of constriction. It contains mucus, perhaps mucopus, and not infrequently a concretion. One or more ulcerative areas appear upon the surface of the mucous membrane; or it is smooth and glazed, the epithelium being displaced by embryonal tissue. The glandular structure, lymphoid structure, and muscular wall are succulent and edematous, if the process is comparatively active; or their true nature is destroyed by cellular infiltration and cicatricial changes. This makes the appendix rigid. The serosa is

dulled and thickened, and plastic peritonitis has resulted in acquired adhesions, binding the organ in a fixed position. Occasionally the enlarged and rigid appendix stands apart, untrammeled by adhesions. The essential features in the pathological process are the alteration in the lumen of the appendix, occlusion of its cecal communication, destruction of its peristaltic power, and the gradual conversion of the appendix into cicatricial tissue poorly vascularized. If perchance such an appendix still retains a communication with the cecum, additional infection is constantly supplied from that channel. Incarcerated concretions are constant sources of mechanical irritation.

The pathological findings explain the recurrent or relapsing attacks that these patients have. In the early stages of this disease, before there is entire destruction of the muscular power of the appendix, efforts to expel the mucus or the concretion explain the attacks of colic; and the persistent soreness with exacerbations is easily accounted for by the associate localized peritonitis.

(b) *Obliterative:* In March, 1894, Senn, with his usual thoroughness, described this pathological change in the appendix, and gave a symptomatology which he considers diagnostic. The preceding year, however, Ribbert contributed a study upon the same pathological subject. Various authors who have described *appendicitis obliterans* show confusion as to the true pathological findings. Ribbert emphasizes the obliteration of the canal, with changes in the mucous and lymphoid structures. Senn seems not to have met with a case in which the entire appendiceal structure revealed obliterative changes. I find numerous cases reported in which the lumen was closed and the walls hypertrophied. I conceive *appendicitis obliterans* to be an inflammatory retrograde change, increasing in frequency with advancing age, resulting in partial or complete conversion into connective tissue of all the structures composing the appendix, with partial or complete closure of its lumen. Bierhoff, Fitz, and Senn attribute it to former inflammation, while Ribbert regards it as a process of involution or retrogression of the organ. We must say that the inflammatory theory is more in accord with our ideas. It may follow the hyperplastic form.

The obliterated appendix to the unaided eye may appear as a slender cord of the usual length of the appendix, found imbedded

in adhesions; or it may appear as a bud from the cecum. Small sacculations are sometimes found, usually at the distal end; mucous inclusions, generally; Ribbert found concretions in 10 per cent. of the cases. Microscopically we find the disappearance of the mucous and lymphoid structures of the appendix; if the process is arrested at this stage the appendix retains its usual length and appears as a tense cord. When the inflammation invades the submucous and muscular layers, converting them into granulation-tissue, which subsequently cicatrizes, the appendix is shortened and indurated. It is in this latter type that we find plastic peritonitis.

II. **Suppurative Appendicitis:** Pus-formation is to be anticipated as the natural consequence of acute infection of the appendix. That the inflammation may terminate short of this, the varieties described show (*vide supra*). With the infliction of a traumatism, that is, the creation of an infective *atrium*, the conditions are all so favorable for the extension of the inflammation that nothing is required for the formation of pus but pyogenic microörganisms of sufficient potency. An infection of sufficient intensity to produce pus in any part of the appendix will be attended by an inflammation of greater or less degree throughout the entire organ and its meso-appendix, although the actual pus-formation may be restricted to a limited area, which corresponds usually to the site of the infection. The duration and intensity of the attack define the limits of the suppuration. Remembering that all forms of appendicitis originate primarily in the mucous membrane, clinically we cannot say to what extent the suppuration has progressed, but we find the following pathological varieties.

1. **Intratubular Suppuration:** A virulent infection of the mucous and lymphoid layers of the appendix results in great edema of the soft structures, and in closure of the proximal end; and cellular destruction by the toxines, assisted by the constriction of the outer layers, is very likely to occur. In such instances the process is usually acute and is not arrested; but occasionally the local resistance is great and the infection is exhausted. We then have within the lumen of the tube a collection of pus limited by the infiltrated and condensed outer walls; and occasionally there are found minute abscesses in the lymphoid tissue, which open

into the lumen and further add to its purulent contents. The limited secretion and the liquefying organic elements increase this accumulation, so that the appendix may be greatly distended with pus. Such an appendicitis may pursue a somewhat chronic course, but at all times presents local symptoms. The peritoneal coat is usually involved, as is shown by adhesions. This is not always so; I have seen an ounce of pus in the appendix without involvement of the serosa. I know that we can have appendicitis without peritonitis, Mr. Treves to the contrary notwithstanding. Such intratubular collections of pus may occupy the entire lumen, or may be sacculated at the distal end. Such a limitation to the pus-formation as the above is unusual.

2. **Intramural Suppuration:** The infection once admitted to the lymph-channels penetrates the submucosa and muscularis, finds its way between the planes of tissues composing the appendicular wall, reaches the peritoneal coat, and there, perchance, peritoneal phagocytosis may arrest it. Dense adhesions are formed, circumscribing and enwrapping the appendix. Visceral and parietal attachments supply additional circulation to antagonize the destructive tendency, and thus for a time the infection is held in check, too late, however, to have prevented the formation of pus within the lumen and in the wall of the appendix. Such an intramural suppuration is usually diffuse; occasionally, however, one or more mural abscesses are formed. Such a pause in the progress of the suppuration can be only for a time. Ulceration and perforation into the peritoneal cavity, or gangrene, may at any moment supervene.

3. **Peri-appendicular Suppuration:** More frequently, however, such a general inflammation of the appendix overcomes the peritoneal resistance, and without any perceptible opening the suppurative process extends to the peritoneum; usually this is accomplished in such a gradual way that nature has time to throw around the advancing suppuration protecting adhesions. The limiting wall is composed of agglutinated intestines, adherent omentum, and anterior or posterior parietes according to the site of the appendix. The location of the appendix influences greatly the possibilities of circumscribing adhesions. Sometimes gradually, sometimes rapidly, pus-elaboration goes on. The accumulation may be relieved by the accidents of perforation into some one of the

hollow viscera with a partial discharge of the pus, or it may burrow and open externally. In either event a reoccurrence of the condition is to be expected. A frequent and much more common termination of these periappendicular abscesses is general peritoneal infection by progressive invasion or by leakage.

The tumor found in suppurative appendicitis does not indicate the amount of pus formed. The elements composing the tumor are the thickened wall of the appendix itself, the contents of the lumen, the thickened meso-appendix, the fibrinous exudates, the enmassed intestines, the adherent omentum, and finally the enclosed pus. And while we have attempted to show that three varieties of non-perforative suppurative appendicitis are found separately, it is most usual to find the three in one.

III. **Perforative Appendicitis**: This form is the result of ulceration confined to a limited area of the appendix, penetrating the entire thickness of the wall, and establishing a fistulous communication between the lumen of the appendix and the peritoneal cavity. Perforation may occur in the first attack of appendicitis, but this is exceptional. The pathological findings in perforative appendicitis indicate the existence of previous disease. A stricture is usually found above the perforation, too well-organized to be of recent development. Concretions, which we have shown in the etiology to be effects rather than causes of appendicitis, are frequently found at the site of perforation or above it. The essential factors of the ulceration leading to perforation are, of course, the microörganisms and their toxines, but mechanical conditions, acting traumatically, play a very important part. Strangulation of the circulation by kinks and torsion, pressure and obstruction to circulation by concretions, occlusion of the lumen by stricture, and external injuries, all afford conditions for the initial pathological changes. The acuteness of the ulcerative process is influenced largely by these predisposing factors. We have no way of telling accurately the time required to accomplish perforation. An ulcer involving all the coats except the serosa may have existed without symptoms for some time, behaving in this way like a latent ulcer of the stomach; and then suddenly and without warning the symptoms of perforation occur. In a general way it may be said that if the appendix is free, if there is no protective peritonitis, the course of ulceration

leading to perforation has been rapid. The presence of adhesions denotes either a previous appendiceal inflammation or a slowly progressing ulceration.

Multiple ulceration of the appendix is very happily explained by Morris as due to necrosis, the result of proliferating endarteritis, the sites of the ulcerations occurring at the ends of the arterial twigs that are first obliterated; this is an infrequent pathological finding.

If perforation takes place in an area surrounded by adhesions formed by previous peri-appendicitis, the noxious elements escaping into this limited space form a peri-appendicular suppuration or circumscribed abscess, the course and events of which are similar to that described under suppurative peri-appendicitis. Perforation occurring without adhesive peritonitis establishes a communication with the general peritoneal cavity; nature may summon her forces to the rescue, and hold within bounds the advancing infection. Acute perforative appendicitis usually pursues, however, a more disastrous course; in a few hours after the occurrence of the perforation the infection has spread rapidly throughout the peritoneum, and we have resulting a general peritonitis.

IV. **Gangrenous Appendicitis:** This may occur primarily, but usually it follows upon a virulent infection of a previously diseased organ. The intensity of the infection may in itself be sufficiently severe to obliterate the lymphatics, obstruct the appendicular artery, plug the venous channels, and thus produce acute gangrene. The presence of a calculus or concretion, or a volvulus or sudden dislocation of the appendix, are often found to explain in part the gangrenous process. Hawkins doubts the existence of either thrombosis or endarteritis. Gangrenous appendicitis is essentially an acute process; no attempt is made on the part of nature to limit it. Death of the appendix in its entirety, or in part only, may occur. Twice I have seen it entirely separated from the cecum, leaving only a round opening to show its former attachment. The gangrenous portion appears as a bluish-black flaccid mass. The offensive odor and the small accumulation of a brownish fluid are the only evidences of the septic peritonitis which in a few hours becomes diffuse, and the patient dies in a very short time, overwhelmed with sepsis.

Such is the behavior of the acute gangrenous process, when it occurs in an appendix previously healthy, or in one not isolated by adhesions. Occasionally we find, either at operation or post-mortem, a gangrenous appendix lying in an abscess cavity. Here perforative or suppurative appendicitis precedes the actual gangrene, and the process is sufficiently slow to permit adhesions to form. The clinical features of such a pathological state are distinguishable from the more acute or fulminating type first described.

COMPLICATING PATHOLOGY.—**General septicemia,** with localized infection of the various tissues and organs of the body, is the most common complication of appendicitis. Common expressions of this complication are seen in septic arthritis, endarteritis, meningitis, pyemic abscesses, and infarctive pneumonia.

Peritonitis, while essentially a septic condition, is such a distinctive inflammation that it stands apart from general septicemia, occurring often from direct infection, and appearing in its several grades and varieties from the most acute diffuse septic peritonitis to the mild fibrinoplastic form.

Phlebitis of the subhepatic, portal, and crural veins, leading to thrombosis or multiple infection of viscera, is a complication found in the course of all grades of appendicitis. Unfortunately it is also a post-operative complication which may arise after the most aseptic technic even in quiescent cases (McBurney).

Cellulitis, especially in the ileac fossa, lumbar and pelvic regions is a complication attending suppurative, perforative or gangrenous appendicitis. It usually arises from implantation of the appendix, partially or entirely, behind the peritoneum, and consequent direct innoculation of the subperitoneal cellular tissue. An abscess so formed is not infrequently attributed to infection from other sources, such as the kidney.

Subdiaphragmatic abscess, occurring as the result of lymphangitis, the burrowing of pus extraperitoneally along the cecum, or from high situation of the appendix, is a complication of undoubtedly greater frequency than is appreciated.

Pleurisy, with or without subdiaphragmatic abscess, is an occasional complication which often passes unperceived. Wolbrecht collects 34 cases.

Internal hemorrhage from ulceration of small arteries, but especially from opening of the iliac vein, is a rare but reported complication.

Ovaritis and **salpingitis** are complications much dwelt upon of late, especially by Price, Edebohls, and others. As we shall show later on, in these cases it is difficult to determine the initial pathological condition.

Nephritis is a complication of great prognostic value.

Cystitis, by contigual infection, is an annoying and often misleading complication.

Fistulous communication with viscera is found. Keen reports a most interesting case in which the communication was with the urinary bladder.

Hepatic suppuration, multiple, from infection through the portal vein, is a fatal complication. In 257 cases of appendicitis Fitz found 11 of suppurative phlebitis and hepatitis; in 100 appendicular post-mortems Einhorn found 6 complicated by pylephlebitis and secondary abscess of the liver.

Intestinal strangulation by bands from local peritonitis of appendicular origin is a complication to be apprehended in any case of latent or so-called medically-cured cases of appendicitis. I have seen complete strangulation of the ileum by the appendix.

Digestive disorders, manifest by painful digestion, flatulency, and constipation, appear in the history of every case of neglected appendicitis.

SYMPTOMS.—In the literature of appendicitis there are many terms used descriptive of the symptoms. According to the course we have *acute* and *chronic* appendicitis; in using these terms, we shall restrict them to this application. Chronic appendicitis is described as being either *recurrent* or *relapsing* in character. Some confusion exists concerning the use of the terms *recurrent* and *relapsing*. McBurney, Hartley, and many others define relapsing appendicitis as a condition of chronic appendicitis with acute attacks separated by more or less long intervals, during which the patient is free from symptoms. In recurrent appendicitis they say that there are the same acute attacks closer together, and that at no time is a patient in a state of health or free from symptoms. Fowler, and Beck make a different and, I think, correct use of these terms. In *relapsing* appendicitis there is no free interval

between the acute attacks. In *recurrent* appendicitis the patient may be symptomatically well between attacks.

A clinical outline of a case of appendicitis to its final fatal termination will give us a comprehensive idea of the general symptomatology. Our patient, usually an adolescent with previous good health, or with insignificant and overlooked digestive disorder, is suddenly seized with acute abdominal pain, paroxysmal in character, and increasing in severity, referred to the umbilical or epigastric regions at first, but soon localized in the right iliac region. The onset of pain is usually attended by nausea and vomiting, which generally subsides when the contents of the stomach have been ejected. Symptoms of shock of variable degree accompany the acute seizure, as is recognized in the elevation of the pulse and the other usual phenomena. Abdominal rigidity, either general or confined to the right rectus muscle, now appears, and with it marked local tenderness in the appendiceal region. Within a few hours the intensity of the pain subsides, its paroxysms occur at longer intervals, its character changes from the acute lancinating or griping to a burning, aching pain, of which the patient is more tolerant. Soreness and tenderness are now distinctive features. Muscular rigidity becomes more pronounced. With the progress of infection the pulse quickens and the temperature rises, constipation becomes a marked feature, rigidity gradually yields to advancing tympany, the stomach is irritable, and vomiting occurs when anything is taken; vomiting is not, however, in this stage a conspicuous symptom. A chill with rise of temperature and pulse, indicating suppuration, adds to the gravity of the scene. The patient's general appearance, his evident anxiety, his dorsal or right lateral decubitus with one or both limbs flexed, attract our attention. By the third day, rarely earlier, a palpable tumor appears, indefinite in outline, but fixed in the appendiceal region.

This tumor is usually described as sausage- or egg-shaped. It may be an indurated cake with flat upper surface. At first its borders are not distinctly defined. Exquisite sensitiveness, muscular rigidity or tympany render palpation difficult. For fear of harmful violence only gentle manipulation must be employed. The mass is immovable. In the early stage of formation, even before we can

outline a distinct tumor, percussion yields a dull note. The value of percussion dullness is not sufficiently emphasized. The position of the appendix frequently covered by the cæcum and small intestine obscures this dullness on superficial percussion, but, if deep pressure is made with the pleximetric fingers, a slight stroke will bring out the deep dullness.

The course now depends upon the accidents of complications. The prostration of the patient, his appearance of serious illness, the hourly increase in the frequency of the pulse, the uncertain and misleading temperature, the persistent constipation, advancing tympany, and the reoccurrence of vomiting, which now becomes frequent and regurgitant, are general symptoms showing unmistakable peritonitis, from which the patient dies—usually from the third to the seventh day. Should he escape peritonitis, the usual cause of early death, he lingers in sepsis, and finally dies from exhaustion or from the development of some one of the many complications.

While the inflammatory process is still confined to the appendix, or has only as yet involved a strictly localized area of peritoneum around it, there are no clinical evidences that are reliable in determining the pathological changes that have occurred in the appendix, and particularly is it impossible to foretell what changes will occur within the next few hours. Even with the development of generalized peritonitis the exact appendiceal pathology will often be in doubt. We may say in a general way that the more fulminant the symptoms, the more serious the pathology, but the most skilled diagnostician is apt to be deceived on either side of the question. After the subsidence of the inflammation, or after it has progressed to the death of the patient, it is possible, looking back over the course of the disease, to make a shrewd guess at what must have been the pathological changes in the appendix itself. However, to attempt to determine this during the active stage of the inflammation, with an eye to adapting special therapeutics to special pathology, is to invite disaster.

SIGNIFICANCE OF SYMPTOMS.—The general symptoms of appendicitis fully developed in its several forms, *pain, localized tenderness, rigidity, vomiting, constipation, formation of a tumor, collapse, pulse* and *temperature,* and *leucocytosis,* are accorded different significance or value in the minds of various surgeons. To accord to each its

diagnostic and prognostic significance, we shall briefly review them.

Pain: This most constant and conspicuous of all the symptoms of appendicitis is influenced largely in its manifestation by the idiosyncrasy of the individual. Distinctly localized pain means a local infection. A general pain, especially if it occurs some hours after the onset of the attack, means a wide-spread infection. A sudden cessation of the pain points either to gangrene or to perforation. In resolution the pain gradually subsides.

Point-Tenderness: Upon the authority of Treves, Pepper, Bryant, and a few others, it is now somewhat fashionable to discredit the value of McBurney's point of tenderness. This, as any other subjective symptom, may be misleading, but when associated with pain, muscular rigidity, and especially a palpably enlarged appendix, it is conclusively diagnostic. It can not be ascertained in some forms of gangrenous appendicitis. It is lost when there are large localized collections of pus.

Muscular Rigidity: Circumscribed muscular rigidity was observed by Shrady 120 times in 300 cases. In acute forms of appendicitis it is more frequently present. In the persistency of the rigidity, as observed at several examinations, consists its value. When rigidity yields and the abdomen is flaccid, convalescence is assured; but when it is displaced by tympany it indicates peritoneal inflammation.

Vomiting and **constipation** or **diarrhea** in the beginning of the attack are inconsequential symptoms. If other symptoms persist and vomiting recurs on the third or fourth day, it must be accepted as an ill omen. Obstinate constipation, with advancing tympany and frequent pulse, shows intestinal paresis due to peritonitis.

Tumor: The development of a tumor is at once bad and favorable. It is bad because it shows that the inflammation has extended beyond the appendix, and has involved the peritoneum. It is favorable in that it reveals the power of nature to control within a definite area the infection. The location of the tumor has a distinct bearing upon the prognosis in the operative treatment. A tumor lying well inward near the middle line, or in the pelvis, cannot be reached or so efficiently drained as one lying in the iliac fossa or loin. Appendiceal tumors formed in cases

in which the appendix is abnormally long, or in which there are abnormalities in size and position of the cecum and appendix, present great difficulties in diagnosis.

Collapse: This can only mean grave infection not only of the appendix, but of the peritoneum as well. In patients very susceptible to pain, shock may appear in the mildest form of appendicitis.

Pulse and **Temperature**: In the symptomatology of the various types, we have referred to the pulse in a vague and rather indefinite way because it is not in accord with other symptoms, nor does it evince the same disturbance in any definite pathological process. A very rapid pulse in the outset usually means a grave infection. A progressively increasing pulse within the first twenty-four hours signifies advancing infection. A sudden increase in the pulse in the second, third, or fourth day of the disease is an alarming symptom. Rapid pulse with tympany and vomiting strongly points to peritonitis. The gravest condition may be present with pulse under 100. I have seen in three cases perforation with septic peritonitis with a pulse of 90.

High temperature in the first twenty-four hours with marked local symptoms means severe infection. "Too much stress must not be laid on the temperature, as recovery may follow a temperature of 105°, and death occur with one nearly normal" (Richardson). "The temperature is absolutely no guide in determining the severity of an attack" (Lyons). A sudden fall in the temperature with increase in the frequency of the pulse indicates perforation.

Leucocytosis: Leucocytosis with the presence of a tumor indicates suppuration. Very high leucocytosis, 20,000 per c.mm., early in the attack indicates peritonitis. A low blood-count, showing less than 2,000 per c.mm., with severe abdominal symptoms and rapid pulse, indicates peritonitis with a bad prognosis.

Jaundice: In neglected cases of suppurative appendicitis jaundice is a grave symptom. If it is associated with recurring chills and fluctuating temperature, pylephlebitis with multiple abscess of the liver is almost certainly present.

Albuminuria: This should influence our operative prognosis.

The General Appearance of the Patient: It is difficult to express the significance of this sign—we do not all read it alike.

As stated in the special symptomatology, the individual symptoms may not be severe, yet the ill look of the patient shadows our prognosis.

ATYPICAL APPENDICITIS.—The appendix may occupy an abnormal position; it may lie entirely extra-peritoneally under the cecum (Bryant and Manley). I have also seen this in several cases. In these cases suppuration of the appendix may lead to infection of the cellular tissue, giving a retroperitoneal abscess, which may burrow upward and become perinephric or subdiaphragmatic in character; or downward into the pelvis, forming a perirectal suppuration. The unusual length of the appendix may lead to local symptoms in other than the iliac or so-called appendiceal region. Malformations, especially an undescended and imperfectly formed cecum, and transposition of viscera, in which the appendix is found upon the left side, are rare occurrences, making diagnosis practically impossible, and greatly confusing the operator. It is not an unusual thing to meet with appendicitis developing in a hernial sac.

Appendicitis, in the symptoms presented, may be atypical. The initial pain, that we have emphasized as the most important and constant of all symptoms, is sometimes wanting. Perforation and gangrene may occur without pain. The absence of circumscribed rigidity and of point-tenderness has been noticed in many cases. Rigidity is a transient symptom and may escape observation; it is very difficult to appreciate it in obese patients. Schrady found it present in 120 out of 300 cases. My experience justifies me in regarding it as a far more constant symptom than these figures would imply. McBurney's point of tenderness, it is true, is present or feigned in many other conditions, but in appendicitis it is rarely absent except in those conditions of malposition of the cecum and appendix. It is quite well understood from our pathology and symptomatology that a tumor is by no means a constant phenomenon in appendicitis. In atypical cases of appendicitis the tumor may be quite remote from its wonted locality: it is found in the pelvis, near the median line, or to the opposite side; close under the liver, or above the crests of the ilia in the lumbar regions.

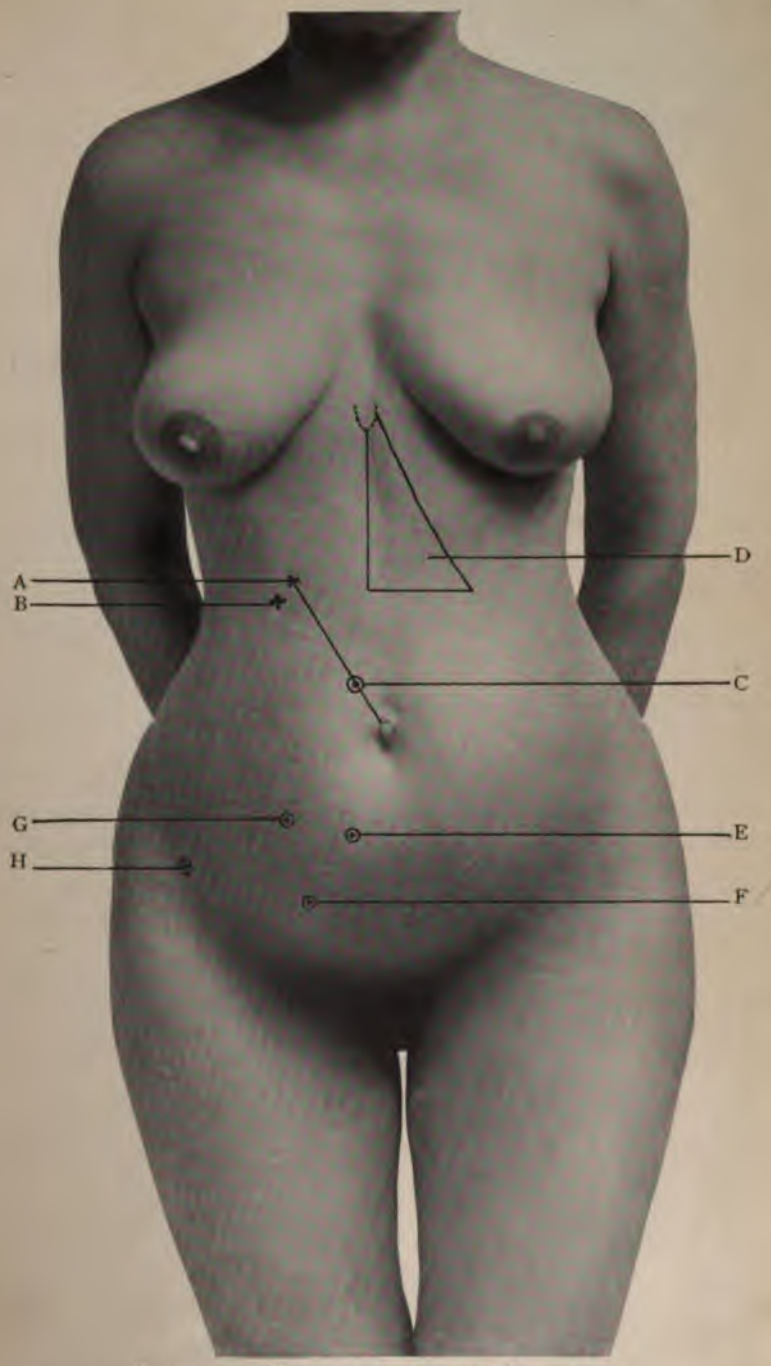
DIFFERENTIAL DIAGNOSIS.—The diagnosis of appendicitis is usually clearly marked by its distinctive symptoms, yet there are

many diseases which present general features so closely resembling that we should in every individual case take them under consideration, and eliminate their possibility. Mere functional disturbances of other viscera confuse the diagnosis. Menstrual disorders, vesical irritations, intestinal disturbances, the nervous perturbances in neurotics, are phenomena that may occur coincident with an attack of appendicitis and obscure the essential symptoms. Through the daily press the public has become acquainted with the symptoms of appendicitis, and imaginative hypochondriacs are frequently assuming the symptoms.

In these minor ailments and nervous conditions the essential symptoms and physical signs of appendicitis are wanting, and patient investigation will usually exonerate the appendix.

Perforating Gastric and Duodenal Ulcer: Certain grave intra-abdominal conditions present symptoms so closely resembling appendicitis that we must resort to every differential aid to show the distinction. In both appendicitis and perforating gastric and duodenal ulcer the onset is usually sudden; in both the pain may at first be diffuse over the abdomen, with general rigidity. Shock is more pronounced and persistent in perforation of the stomach and duodenum. Constipation occurs in both. Meteorism is early and conspicuous in perforating gastric ulcer—it develops in six to twelve hours; it is a late sign in appendicitis. With the onset of appendicitis there is usually elevation of the pulse and temperature. The presence of fever is one of the most important differential signs, distinguishing appendicitis from a chronic visceral ulcer with perforation. In a general way the symptoms of gastric and duodenal ulcer are directed to the upper zone of the abdomen, while those of appendicitis are below the umbilicus. In gastric and duodenal ulcer, and cholecystitis, especially the last, the escaping visceral contents directed by the colon may make their way to the iliac fossa, and hence it is that pain is often felt in that region; it is a secondary, and not the initial pain, however. And so we may have a tenderness in the iliac region in duodenal ulcer, yet it is not that specific point-tenderness of appendicitis to which we ascribe the greatest value.

PLATE IV.



SIGNIFICANT POINTS OF ABDOMINAL TENDERNESS.

A. Tip of ninth rib.
B. Tip of tenth rib, the point of usually assigned gall-bladder tenderness.
C. Robson's point of tenderness in cholecystitis.
D. Head's triangle, area of tenderness in gastric ulcer.
E. Point of tenderness in ureteritis.
F. Point of tenderness in ovaritis.
G. McBurney's point, in appendicitis.
H. Anterior spine of ileum.

Appendicitis.	*Perforating Gastric and Duodenal Ulcer.*
History.—May be wanting, or there may have been similar previous attacks.	Usually history of gastric disorder for some time.
Age.—Any age, most common from 10 to 30 years.	Usually between 20 and 40 years.
Sex.—More frequent in males.	Far more frequent in females.
Vomiting.—Occurs in about one-half the cases.	Only occasionally occurs at the time of perforation. Blood may be found in vomitus and stools.
Pain.—Of greatest intensity in the iliac region.	Of greatest intensity in the epigastrium, right and left hypochondria.
Tenderness.—McBurney's point of tenderness in the right iliac region.	More diffuse, in the epigastric and hypochondriac regions. Iliac region may show sensitiveness, but no definite point-tenderness.
Muscular Rigidity.—Right rectus and right external oblique are especially rigid immediately over the appendix.	Rigidity of the muscles in the epigastric region most marked.
Pulse and Temperature.—Frequent pulse and elevation of temperature are early symptoms, present throughout the attack.	Afebrile in onset. Rapid pulse and low temperature follow perforation. Late rise of temperature due to peritonitis.
Shock.—Accompanies only the perforative or gangrenous form.	Early and pronounced.
Tumor.—Usually detected by the third day, in the right iliac region.	No tumor; occasionally induration from peritoneal adhesions at site of ulcer.
Effacement of Hepatic Dulness.—Rarely observed in appendicitis.	An early phenomenon in gastric perforation.

Intestinal obstruction, especially *intussusception,* may be mistaken for appendicitis. The vomiting, the localized pain in the iliac region, the tenderness, and relative constipation, with the development of a tumor, are features in common. In intussusception the pain is more paroxysmal in character, and in the interval the patient is more comfortable; fever is usually absent, never high until the onset of peritonitis; muscular rigidity is not marked, and is generally entirely absent; the tumor itself is soft, of different shape, of more distinct outlines, and changes in position with the progress of the intussusception; tenesmus with mucohemorrhagic stools is in itself almost sufficient to eliminate appendicitis; rectal examination may detect the apex of the intussusceptum.

Typhoid Fever: The iliac tenderness which is sometimes marked in the early stage of typhoid fever may lead this disease to be mistaken for appendicitis. This confusion is more real than one might imagine, yet the difference in onset and in the physical phenomena of the two diseases is very wide. Typhoid fever, with its feeling of lassitude, headache, anorexia, coated tongue, epistaxis, and diarrhea, is certainly distinctive from appendicitis, and the only features in the early stage of typhoid that resemble appendicitis are local iliac tenderness and vomiting; but there is absence of rigidity, the absence of a tumor, the negative results of a rectal examination; the development of an eruption, the enlargement of the spleen; and by the end of the first week the temperature curve and Widal's reaction should establish the diagnosis of typhoid fever.

It is in the later stage of typhoid that a combination of circumstances may exist which would lead to the erroneous diagnosis of appendicitis. If typhoid has not been positively diagnosed, if it happens to be an ambulatory case, if localized peritonitis precedes the perforation of typhoid ulcer, and if the ulcer is in the cecum or appendix, or if the case is not seen until the perforation takes place, the difficulty of determining its nature must be apparent. Happily the surgical indications are the same in both instances.

A **movable kidney** will not infrequently descend to the region of the appendix, and here by traction upon and constriction of its blood-vessels and ureter acute symptoms of abdominal pain

with vomiting, serious shock, and local tenderness may appear. The kidney in its abnormal position may be mistaken for the inflammatory tumor of appendicitis. We must keep before us the possibility of movable kidney, especially in neurotic females. Palpation will reveal the absence of the kidney in its normal position; the kidney-shape of the tumor, and the possibility of returning it to its normal position when the patient assumes the dorsal posture, make clear the diagnosis. The complexus of symptoms grouped by Dietl and constituting a crisis is often misinterpreted as an attack of recurrent or relapsing appendicitis. Careful palpation in the interval should establish the diagnosis. We must bear in mind the frequent association of the two conditions.

Renal colic with its violent onset, its paroxysmal pains radiating downward through the lumbar and iliac regions, with its attendant vomiting, great depression, and spasm of the abdominal muscles, may be mistaken for appendicitis. However, the urinary symptoms, retraction of the testicle, the pain reflected to the genitals, the absence of persistent rigidity and definite point-tenderness, and the course and short duration of the attacks determine the diagnosis.

Cystitis: Occasionally, as we have found, the appendix lies in the true pelvis, and its distal end may become attached to the bladder and give rise to symptoms resembling cystitis. The absence of blood, mucus, and pus from the urine, the sudden onset, the iliac pain and tenderness, rectal and vaginal examination should exclude vesical inflammation.

Salpingitis and **ovaritis** pursuing an acute course may give rise to such pronounced abdominal symptoms as to obscure the pelvic origin of the disease. Right pyosalpinx or right ovarian abscess may be mistaken for appendicitis, but the knowledge of the preceding uterine inflammation due to puerperal, specific, or traumatic infection, the history of menstrual disorders, the chronic character of the disease, and the intimate connection that the enlarged appendages have with the uterus, are guiding differential points; but the essential point in the differentiation is the absence of the three cardinal symptoms of appendicitis, namely, sudden onset of pain, McBurney's point of tenderness, and muscular rigidity. A large pyosalpinx or ovarian abscess may form a tumor occupying the iliac fossa, resembling peri-appendicular

suppuration. Even in such instances the history and the manner of the development of the tumor should make the diagnosis.

Ruptured Ectopic Gestation: It would appear that the history and physical signs and the accompanying menstrual disorder should distinguish a ruptured ectopic gestation from appendicitis. In both there may be amenorrhea; in both violent pain attends the onset; in both muscular rigidity may be present, and shock is a constant symptom. In three instances, as previously stated, I have seen an attack of appendicitis bring on uterine hemorrhage. In one of these cases there was amenorrhea for eight weeks, then hemorrhage was profuse. Vaginal examination was negative, yet an erroneous diagnosis of extra-uterine pregnancy was made. The appendix was found gangrenous and entirely separated from the cecum and an opening as large as a dime marked its former site of attachment; through this opening intestinal contents had abundantly escaped into the peritoneal cavity. The patient was submitted to operation in a little less than six hours after the occurrence of the first symptom, yet there were evidences of general peritonitis. Copious irrigation with drainage through each loin and vagina established successful sewerage, and the patient recovered. Happily such confounding conditions will not often occur. The general symptoms of pregnancy, the presence of a tumor, the lateral or forward displacement of the uterus, and at the time of rupture the recurring syncope, the presence of free fluid within the abdomen or the circumscribed broad-ligament tumor, should make the diagnosis.

Torsion of Ovarian Pedicle: Torsion of the pedicle of a small, unrecognized ovarian tumor may cause it suddenly to enlarge and to become visible and palpable. The accompanying symptoms of pain, shock, nausea, and vomiting suggest appendicitis; yet the character of the tumor, its tense elastic feel, its pelvic origin, its rapid development, and the diffuse tenderness eliminate appendicitis.

Nicholson, Richardson, Mixter, and Darnall report cases of gangrene of Meckel's diverticulum mistaken for appendicitis.

PROGNOSIS.—Appendicitis in infancy is prone to pursue a very destructive course. Peritonitis quickly develops. Very obese patients, alcoholics, and rheumatics offer poor resistance, and frequently succumb in a short time to the ravages of infection;

and if subjected to operation, they are not favorable subjects. In a general way it may be said that the prognosis is influenced by the severity of the initial symptoms; the exceptions to this rule are so numerous that we think a better guide is the persistency of the symptoms. Our confessed inability always to interpret the symptoms as exemplifying the different pathological types must necessarily obscure the prognosis. The known liability to the sudden transition of a mild or chronic form into the severest types by the accidents of perforation and gangrene precludes a favorable prognosis even in the simplest cases. It is variously estimated that from 7 per cent. to 10 per cent. of patients die from the first attack. What proportion of the remaining 90 per cent. have recurrence must necessarily remain an unknown quantity. After two attacks, says McBurney, a patient is sure to have a third, the gravity of the condition increasing with each renewal. The symptoms in the first 18 to 24 hours clearly establish the diagnosis and probable course of the disease.

In cases of relapsing or recurrent appendicitis, if the attack comes on gradually, with definite local symptoms, the probability is that it will subside, as upon previous occasions; but if the attack is ushered in by pronounced symptoms—especially diffuse pain, general rigidity, and shock—perforation or gangrene may be very properly feared. Temperature in the first 24 hours may reach 104° or 105°, and yet the patient may recover. A sudden rise in the temperature after the third or fourth day of the disease usually means circumscribed peri-appendicular suppuration. The knowledge that gangrenous appendicitis and septic peritonitis may exist with practically a normal temperature leads us to place but small reliance upon prognostic value of the temperature curve. A pulse of increasing frequency indicates advancing sepsis, and while serious pathological changes may occur without marked circulatory disturbances, much reliance may be placed in the behavior of the pulse. Constipation and tympany not due to opium are ugly symptoms; if they are accompanied by vomiting and a fast and failing pulse, a fatal termination may be almost certainly predicted. The development of a tumor has an immediate, but not an ultimate bearing upon the prognosis. It assures for a very limited time comparative safety. If the general symp-

toms are persistent and progressive with the growth of the tumor, the condition is exceedingly critical. If the tumor is circumscribed and the patient is manifestly better, it is probable that resolution will take place to a very great degree. Severe symptoms in the absence of a tumor are of the gloomiest possible portent. Epigastric distention, due to acute paralytic distention of the stomach, without general meteorism, is sometimes one of the first symptoms of diffuse peritonitis.

The development of an acute nephritis is one of the gravest complications, and materially affects the operative mortality.

TREATMENT.—**Medical:** "There is no medical treatment of appendicitis" (Osler). With this statement from so high an authority, according as it does with my own convictions, I may proceed to the surgical management.

Surgical: The impossibility of determining clinically the pathological changes going on in the appendix when it is the seat of inflammation is the principal factor in making appendicitis an absolutely surgical condition. If we had any reliable constitutional or local evidence of what was taking place within the abdomen, we might in many cases be able to await the interval before instituting surgical measures.

The treatment of appendicitis, when reduced to first principles, resolves itself into the prevention of peritonitis (and other complications) if possible, and the relief of peritonitis, once it has developed. The appendix itself being functionless can be sacrificed without compunction to attain these ends. If the surgeon will keep in mind the fact that the protection or relief of the peritoneum is his main consideration always, and that complete surgery is of secondary importance, there is no stage of appendicitis with one possible exception that is not surgical, and in which surgery does not offer the greatest safety to the patient. The possible exception to this rule is when the inflammation is already distinctly subsiding, when the case is first seen, and here we encounter the drawback of having to rely upon that very unreliable evidence, the history as obtained from the patient or from friends.

The impossibility of basing surgical indications on appendiceal pathology is shown by the fact that no authority attempts such a feat. When an attempt is made to vary the treatment, it is usually done on a time basis. When, however, we look squarely at this

plan of measuring appendicitis for purpose of treatment with a twenty-four hour unit, it can be seen at once that it is more or less grotesquely ridiculous. One patient will die within a single period of twenty-four hours, while another will get practically well in the same length of time. Since neither time, symptoms nor signs will guide us in the selection of cases, we must perforce rely upon the general application of surgery, which, when done intelligently, will never add to the gravity of the case, and will at once check the stream of possible complications by drying up the source.

The mortality of operation done while the infection is still confined to the appendix is negligible, while the mortality of operation done in the face of complications is that of the pathology already present. This plan of treatment is based upon the supposition that the operative work is in the hands of a competent, skillful surgeon. If a patient must take his choice between Nature's efforts and blundering surgery, he may well select the former.

For cases that do not require drainage either the McBurney or Battle-Kammerer incision is satisfactory, though the latter can be made to give much more room, and for that reason is more generally applicable. When drainage has to be used in a case in which the Battle-Kammerer incision has been employed, it is best to use a stab wound for this purpose if possible.

In abscess cases the McBurney incision is preferable. We desire here little or no intra-abdominal manipulation and this incision gives ample room for the evacuation of the abscess. The wound infection which is certain to follow is less apt to result in hernia with this incision than with any other. The removal of the appendix when a local abscess is drained should be left for a secondary operation unless it is easily accessible and can be gotten out without breaking up any of the limiting adhesions. The plan advocated by Murphy of opening into the general cavity and carefully packing off before opening the abscess is probably the best if we are determined to drain the abscess and remove the appendix at the same time. However, in the hands of the average surgeon two operations, one for drainage and one to remove the appendix, will in all probability give a lower mortality in these cases.

When a generalized infection of the peritoneum has occurred before operation is done no manipulation of peritoneal surfaces

must be indulged in. The appendix must be removed or sequestrated as promptly as possible and the rest left to Nature's efforts.

CONTRAINDICATIONS TO OPERATION.—In an acute attack, especially if it is the first, if the symptoms are mild and show a tendency to subside in twelve or eighteen hours, operation should be deferred. In relapsing or recurrent appendicitis, if the symptoms are moderate in severity, distinctly localized, we should postpone operation until the interval. If a tumor has formed and shows a tendency to decrease in size, and if there is no marked leucocytosis with subsidence of general symptoms, we should watchfully wait for resolution and operate when the active inflammatory condition has subsided. In wide-spread peritoneal infection it is sometimes better to defer operation than to subject the patient in his intensely septic condition to the danger of operative shock. Operation should never be done upon a patient who is manifestly dying.

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ABSCESS OF THE LIVER.

DEFINITION.—An intracapsular suppurative process involving the parenchyma, bile-ducts, or blood-channels of the liver.

SYNONYMS.—Hepatic abscess, suppurative hepatitis.

FREQUENCY.—Out of 28,034 cases of internal diseases treated at the Medical clinic at Zurich, Dudly found but 12 cases of hepatic abscess, clinically or anatomically recognized. Mejia reports that, in the City of Mexico out of 88,416 deaths, he found that 1,985 were due to hepatitis purulenta. Ewald, from the records of the Berlin Pathological Institute finds hepatic abscess in 1.5 per cent. of all autopsies. Moore, from the records of St. Bartholomew's Hospital, London, finds its relative frequency to all internal diseases less than 1 per cent. Musser places its frequency at nearly 3 per cent. Osler finds it not very infrequent in Baltimore. The average surgeon will meet with from one to four cases in the course of a year in this climate.

ETIOLOGY.—In the literature of this subject we meet with the terms 'spontaneous,' 'primary,' and 'idiopathic' abscess, which create much confusion in its study, and are manifestly confessions of ignorance of the etiology of this affection. That any disease can spontaneously spring into existence without an essential cause, or be idiopathic in origin, is contrary to all observation of progressive medicine; yet we must confess that the active or essential cause is not always demonstrable. Unfortunately there are many instances of abscess of the liver in which the determining cause is not ascertainable. To assign these to such vague influences as thermic changes, alcohol, and malarial cachexia, which have no known pyogenic power, leaves unanswered the question of pathogenesis. Let us confess that in certain forms of tropical abscess the cause cannot be discovered, yet in their pathological and clinical course they present features identical with abscesses in which the etiology is well known. Harley says that every form of hepatic abscess is invariably preceded by a recognizable cause, and that the so-called idiopathic abscess may

be only a metastatic abscess in which the origin has eluded detection. Allbutt accepts the term 'idiopathic' "only in its negative bearing," that is, in those cases in which the cause is unassigned. Neil Macleod maintains that tropical abscess, the only form for which spontaneous evolution is claimed, is the result of dysenteric ulceration.

It is, therefore, not assuming too much for the future to predict that the terms 'spontaneous' and 'idiopathic' will fall into disuse, and we shall attain to greater precision in the pathogenesis of hepatic suppurations.

Climate plays a very important part as a predisposing cause of hepatic abscess. It is a disease of the widest geographical distribution, yet it is comparatively infrequent in temperate regions, causing less than 3 per cent. of the total mortality. It prevails in the tropics endemically, as in tropical Asia, Egypt, and Algiers, where it constitutes 5 per cent. of all diseases.

The disturbances of digestion incident to hot climates impress themselves especially upon the liver, producing hyperemia of that organ. The insufficient and poor quality of food, and the prevalence of malarial toxemia, are additional causes, which Rouis thinks sufficient to excite hepatitis. Foreign residents of these localities are usually free drinkers of alcohol, do not adapt their diet and mode of living to the demands of the climate, and hence are especially liable to the development of hepatic abscess. All authorities ascribe to alcohol an important rôle as an etiological factor. Of 36 cases noted by Sachs, two were in women, one of these a notorious drinker; and all the men were intemperate. Waring gives 300 cases, of which 67.5 per cent. were drunkards.

Malaria has been considered to be a determining cause of hepatic abscess, but now that we know the biological properties of Laveran's hematozoa, and that they do not cause suppuration, the theory of the malarial origin of hepatic abscess should at once be rejected; yet that it does render the hepatic tissues susceptible to infection is very clear, and it must, therefore, be regarded as a predisposing cause (Semmola).

Age: Hepatic abscess is rare in children. Musser has collected 34 cases in children under 15 years of age; many others have since been reported. Multiple abscess is met with in the new-born, from infection through the umbilical vessels.

Sex: Women enjoy almost absolute immunity from hepatic abscess. In Sachs's 36 cases only 2 were women. Rouis's series contained 250 men and 8 women. Of Waring's 300 collected cases but 9 were women. Eichhorst places it at the ratio of 30 males to 1 female.

It would be futile to discuss or seek to untangle the confusion that exists concerning the relation between tropical dysentery and hepatic abscess. In 1845, Budd propounded the doctrine that hepatic suppuration was due to the absorption of purulent matter from intestinal ulceration. Murchison refutes the idea that an open sore in the intestinal tract produces the hepatic suppuration so commonly met with in hot climates, and which he designates 'tropical abscess,' with the fourfold argument: (1) tropical abscess is not uncommon in the absence of all history and symptoms of dysentery; (2) tropical abscess often precedes the occurrence of dysenteric symptoms; (3) patients dying of tropical abscess often show no evidence of intestinal ulceration, active or healed; (4) abscess occurring from ulceration, that is, pyemic in character, is usually multiple, whereas the tropical abscess is usually single, rarely more than three. The investigations of Morehead, with 21 fatal cases of abscess of the liver without any sign of intestinal ulceration; and Waring's series of 204 cases, in one-fourth of which there were no ulcerations, cicatrices, or abrasions in the intestines, strengthen the position of Murchison. Having established the identity of a peculiar solitary tropical abscess, which sometimes existed without discovered intestinal lesion, and which Martin, Morehead, Frerichs, and many later observers ascribe to heat, moisture, and alcohol—intangible causes—it therefore naturally followed that such an abscess was designated 'spontaneous,' 'primary,' or 'idiopathic.'

In 1883, Kartulis first called attention to the etiological relation of the ameba to dysentery. In 1887, he described the living motile ameba in the contents of hepatic abscess. Osler, in 1890, reported a case of dysentery with abscess of the liver in which the ameba was found in the stools and in the pus from the abscess. Since then the relation of the ameba to dysentery has been exhaustively studied. There still prevails much doubt as to the part it plays in the etiology of abscess, but this seems to arise from the fact that it is forgotten that in the tropics, as else-

where, dysentery may be due to many causes, and may exist in various forms. That tropical dysentery is frequently complicated with, or followed by abscess of the liver is well known. Rouis found that 90 per cent. of his cases of abscess of the liver had dysentery. In 1,429 autopsies on individuals dying of dysentery Councilman and Lafleur found 306 abscesses of the liver, about 1 in 4. According to Kartulis, in 500 cases of dysentery over 50 per cent. had abscess of the liver. Of 40 cases of amebic dysentery occurring in America, collected by Howard and Hoover, 18 had abscess of the liver. If this parasite is capable of producing the destructive changes in the intestinal wall, it would appear that they are able to accomplish the same effect in the liver. Kelsch and Kiener in their excellent work, "*Traité des maladies des pays chauds,*" 1889, found that of 314 cases of hepatic abscess dysentery coexisted in 268; and the constancy of the presence of the ameba in both affections led them to conclude that abscess of the liver is caused by the ameba coli found in the ulceration of the intestines. Neil Macleod says that dysentery is the invariable precursor of tropical liver-abscess. Forbes says that amebic dysentery is that form after which tropical abscess is found in most cases, if not always. In 80 per cent. of necropsies performed on patients with liver abscess, some one of the following conditions were found: the amebæ coli were in the stools, on the surface of the dysenteric ulcer, or in the wall of the intestine; cicatrices of the intestinal mucous membrane were found; the amebæ were found in the walls of the abscess or in its contents. Councilman and Lafleur regard the ameba the essential morbific agent, capable independently of producing hepatic suppuration.

It is very difficult to ascribe to amebæ their etiological significance. Virchow regarded them only as the carriers of micro-organisms. Zancarol considers hepatic abscess a microörganismal disease, the prime factor of which is the streptococcus, and holds that the ameba plays no rôle in the pathogenesis. Kartulis himself admits the association of other bacteria. Kruse and Pasquale believe that it is the combined action of amebæ and bacteria which produces the destructive changes in the liver.

It appears that sufficient proof is in hand to justify us in classifying amebic dysentery as a separate and distinct disease,

often giving rise to the complication of hepatic abscess. But little weight can be given to the arguments deduced to show that they are simultaneous localizations of the same infective agent, nor is it at all clear that in any definite series of cases the abscess of the liver is the exciting cause of the dysentery, as Annesley asserts, and as Flexner believes occurred in one of his cases.

Pus from hepatic abscess is often sterile. Calmette, of Cochin-China, quoted by Schweiger, found the pus sterile in 7 hepatic abscesses; and Kartulis found it so in 8 out of 13 cases. It is argued that this does not disprove that microörganisms were not once present in pyogenic potency, but that they have disappeared, destroyed by the liver-cells or bile. Zancarol claims that the pus from a hepatic abscess is just as deleterious, whether sterile or not, as from other suppurative organs.

Microörganisms, other than amebæ, play a joint or independent rôle in the pathogenesis of liver-abscess. The streptococcus, the bacillus pyocyaneus, the bacillus fetidus, and the bacterium coli have all been found. Schweiger in several cases found the staphylococcus aureus.

The amebæ reach the liver by the portal circulation. The situation of the ulcer in the colon, the presence of the amebæ on the surface of the ulcer, in the wall of the intestine, and in the venous radicles, explains their easy transmission to the liver. Councilman and Lafleur report a case in which the amebæ were found in the exudate of peritonitis, thus showing the possibility of infection by route of the peritoneum. Evidence is wanting to show that they reach the liver by the bile-channels or by the lymphatics.

In this climate the most frequent cause of abscess of the liver is the transmission of infectious matter to the organ by the circulation from focal suppuration elsewhere. The infection may reach the liver by the portal vein, by the hepatic artery, and by the cystic vein. Of special importance is infection by way of the cystic vein and its branches. The veins of the gall-bladder and bile-ducts are roots of the portal vein; septic stations in their route may give rise to pylephlebitis or septic emboli. The possibility of regurgitation from the vena cava, while suggested, is scarcely plausible. Suppuration in any organ contributing

blood to the portal vein may furnish the infected embolus, which, reaching the liver, will give rise to suppurative hepatitis.

We have attempted to show the causal relation existing between amebic dysentery and amebic abscess. Dysentery in any of its forms, in this climate or in the tropics, may and does occasionally give rise to abscess of the liver, multiple or single. Lesions of the digestive tract, of whatsoever character, owing to the direct vascular connection through the portal vein, are especially prone to initiate the formation of an abscess. This may be metastatic or embolic in character, the embolus being septic, or, as Virchow designates it, malignant. There may be an extension of the inflammation through the vein in the form of pylephlebitis, invariably resulting in fatal suppurative hepatitis.

Suppurative appendicitis ranks second only to amebic dysentery as a cause of secondary abscess of the liver. Of 17 cases of abscess of the liver Jackson found that in 10 the cause was appendicitis. In 527 cases of appendicitis Fitz found 11 of suppurative phlebitis and hepatitis. In 100 appendicular post-mortems Einhorn found 6 complicated by pylephlebitis and secondary abscess of the liver.

Abscess of the liver as a sequel of typhoid ulceration is rare. Bokai, quoted by Walsh, reported one case in which the inflammation was traced along the portal vein. In 2,000 autopsies made in Munich on persons dying of typhoid fever, abscess of the liver was found in 12 only (Osler). Romberg reports 9 cases of hepatic abscess in connection with typhoid fever, and speaks of abscess of the kidney, spleen, and mesenteric glands in association. In the *Medical News,* Nov. 15, 1890, I reported a case of solitary abscess of the liver in a young man, occurring four months after a well-defined case of typhoid fever. After free incision and drainage the patient recovered. This is the only instance I am aware of in which this sequel of typhoid fever has occurred in Nashville. Typhoid infection may cause hepatic abscess in three different ways: (1) it may be metastatic from the intestinal ulcer or some other point of suppuration; (2) from typhoid ulceration of the biliary passages; (3) in consequence of pylephlebitis. That the affection occurs as the result of ascending thrombosis, producing symptoms of peritonitis, may account for the fact that it is often not recognized clinically.

Primary tubercular abscess of the liver is exceedingly rare. Thorowgood and MacKenzie each report a case of undoubted character, and in their researches find it a very exceptional occurrence.

Operations upon the rectum, pelvic floor, and genitals have contributed their quota to the formation of hepatic abscess. Bettelheim reports a case of hepatic abscess of gonorrheal origin; the infection was probably a thrombosis originating in the pudendal plexus. No embolus was found, yet Thierfelder tells us that embolism of branches of the portal vein may occur without leaving any trace.

Pancreatitis, abscess of the spleen, gastric and duodenal ulcers are other intra-abdominal diseases which may supply through the portal vein infection to the liver. Hehir records a case of multiple abscess of the liver from ulcer of the duodenum following a severe burn; the pus contained neither staphylococci, streptococci, nor amebæ, but a number of ordinary small micrococci were found.

While infection by the portal circulation is the most frequent cause of hepatic suppuration, infective wounds and pyogenic diseases in other organs and parts of the body whose venous blood pursues the channels of the greater circulation may incite suppurative hepatitis. In such instances there may be no distinct embolus, but the pyemic blood, with its contained microörganisms, finally reaches the liver, and the bacteria, largely contained within the white corpuscles, produce swelling and disintegration of the liver-cells, and a pus-producing plant is established. An embolus in the general circulation may reach the liver by the hepatic artery after traversing the heart and lungs. Wounds of the head were formerly supposed to be especially prone to be followed by hepatic abscess; it is now known that there is no such special predisposition; the lungs are far more likely to be involved by septic infarctions. In death from surgical diseases and injuries Waldeyer, quoted by Edwards, found that 66⅔ per cent. of the cases had pulmonary abscess, while only 6 per cent. had hepatic abscess.

Gall-stones may create an *atrium* for infection by the intestinal bacteria. The suppurating process may extend to the liver as a cholangeitis, or by inflammation or emboli through the cystic

vein. In two cases of hepatic abscess, the sequel of typhoid fever, suppurative cholangeitis and cholecystitis with gall-stone were found. The bacillus typhosus no doubt initiated the entire pathological process. German writers regard gall-stone, after dysentery, the most common cause of abscess of the liver.

Ascarides entering from the intestine through the common duct may excite a mechanical irritation, which becoming infected leads to the formation of hepatic abscess. Hirschfeld, quoted by Lingen, has collected 50 cases in which ascarides found their way into the bile-ducts and produced purulent processes; most of these were in children. Musser collected 8 cases from the same cause, all in children.

Echinococcus cysts may undergo changes resulting in hepatic suppuration.

Wounds of the liver, open or concealed, may for obvious reasons give rise to suppurative hepatitis; yet such an occurrence is comparatively rare. Musser has been able to collect 8 instances.

PATHOLOGY.— 1. **Amebic Abscess**: In the pathogenesis of hepatic abscess we have accorded to the ameba coli individual importance. Its association with dysentery leaves but little doubt of its etiological relation, producing, in the colon chiefly, a characteristic ulceration, the essential feature of which is that the primary change is in the submucosa, leading to the formation of abscesses, which may open through the mucosa, giving rise to a deep ulcer with overhanging edges. Unopened abscesses surround this ulcer. The amebæ burrow in the submucosa and into the glands, invade the muscularis, and all the coats of the bowel are ultimately involved. Their absorption by lymphatics is in a great measure precluded by their size, hence they reach the liver by the portal circulation. The necrobiotic process there incited is not definitely understood, but here, as in the intestinal ulcer, the destructive process is possibly modified by the action of bacteria accompanying them. We have previously explained why these bacteria are not always found. Whether the destructive process begins in the hepatic-cells, as Ewald seems to think, or, as Lafleur contends, in the interlobular areas, is undetermined. In either event the purulent softening of the affected parts breaks the capsule, and two or more acini

coalesce and gradually involve adjacent acini until the destructive process may involve an entire lobe of the liver or more.

Contents: The contents of the abscess-cavity depend largely upon the duration of the process and the size of the abscess. In very small abscesses the contents are gelatinous; in larger ones, of more fluid consistency. In recent abscesses the contents are brown, chocolate-colored, usually described as resembling anchovy sauce, consisting of liver-cells and detritus; little or no bile is found. In older abscesses yellow or greenish-yellow pus is met with, peculiar in character and not described as laudable. Microscopically examined, the contents will show the amebæ, though they are not always detected in the pus first discharged. The contents are usually sterile of bacteria, though various pyogenic germs may be found. The striking characteristic of the pus from an amebic abscess is the absence of leucocytes. The liver-cells seem to have undergone necrobiosis and liquefaction, and show no leucocytic invasion.

In recent abscesses the wall of the cavity consists of edematous liver-substance, soft, irregular, and with attached masses of necrotic tissue. In large and old abscesses usually three zones can be made out: (1) a necrotic central zone; (2) a highly refractive reticulum; (3) a layer of granulation tissue (Howard and Hoover). There is no evidence of leucocytic reaction around, unless there be other infection. The amebæ are more often found in the walls of the abscess than in its contents. The more acute the abscess, the greater the probability of being able to detect the amebæ. Around the abscess there may be focal areas of non-suppurative necrosis, due to the absorption of a solid toxic substance produced by the amebæ. The liver-tissue surrounding the abscess is usually pale, and does not show evidences of inflammation.

Number: The tropical, or as it should be called, the amebic abscess, is usually single, rarely more than three. Occasionally there is one large abscess with smaller satellites; and after the evacuation of the principal abscess, the smaller ones may attain to large size and pursue a destructive course.

Seat: Dysenteric abscess elects for its development usually the right lobe of the liver. Of 156 cases Rouis found the seat of the disease in 122 to be the right lobe. This is accounted for by the

greater relative size of this portion of the liver. It is further explained by Langenbuch by the distribution of the branches of the portal vein: that branch leading to the right lobe is larger and runs parallel to the main stem, while that going to the left lobe is at right angles to it. The abscess is usually situated near the upper or convex surface of the liver; hence the frequent diaphragmatic and thoracic complications. It must not be inferred that the left lobe or any part of the liver is exempt from this form of suppuration.

Size: An abscess varies in size from that of a small marble to that of a child's head. The entire liver may apparently be converted into a sac. As much as seventeen pints of pus has been found in the cavity of a hepatic abscess.

Appearance of the Liver: Very large abscesses may be concealed within the substance of the right lobe of the liver without giving rise to any enlargement, or otherwise affecting the appearance of the organ. As they approach the surface they may produce distinct elevations, exciting peritoneal irritation, leading to the formation of adhesions to the parietes, diaphragm, or viscera. The complication of diaphragmatic adhesions and inflammation fixes the right wing of the diaphragm and limits its movement in respiration.

2. **Traumatic Abscess:** Following injuries and wounds of the liver, suppuration may occur. In subparietal injuries previous disease of the liver or bile-channels may supply the pyogenic organism. The injured parenchyma and stagnated bile are favorable to infection from the intestine, and as such contusions and lacerations of the liver are often attended by intestinal injuries, it is not difficult to see why suppuration should occasionally occur. In wounds of the liver with an external communication, as gunshot or stab-wounds, the infection is often introduced by injudicious probing. Traumatic abscesses of the liver are usually single, the size depending upon the severity of the infection and the extent of the lesion. They are always septic in character; the contents show an abundance of microörganisms. The pus is yellow, green, or reddish-brown from the admixture with blood and disintegrating liver-tissue. The walls of the abscess are irregular, ragged, and composed of condensed liver-tissue, infiltrated with leucocytes. The local inflammation excites sur-

rounding peritonitis, which may be plastic or suppurative in character.

3. **Embolic Abscess:** The American surgeon occasionally meets with amebic abscess, but usually the form of suppurative hepatitis encountered is embolic or metastatic in character from a septic focus. A thrombosis undergoing "puriform deliquescence" in the ultimate radicles of the portal vein, or in any part thereof, may supply the germ-laden embolus, which, lodging in some of the interlobular ramifications, produces engorgement, liquefaction and purulent degeneration of the parenchyma of the liver. The inauguration of this secondary infection through the channels of the portal vein is readily comprehended. It is not so easy to explain the origin of those abscesses of the liver secondary to infective processes in bones, tissues, and organs in communication only with the general circulation. The frequency of pulmonary infarction with subsequent disintegration in pyemic conditions has been noted; the embolus has only to traverse the right side of the heart, thence to the lungs where it is arrested in the minute capillaries. The experiments of O. Weber years ago established that certain forms of emboli, especially flocculi of pus, pass the pulmonary capillaries without difficulty, enter the left heart, and thence go out to the systemic circulation, and reach the liver and other organs, causing abscess. This explains those cases of peripheral venous thrombosis in which there is abscess of the liver without abscess of the lung. Of course, in such instances the hepatic artery carries the embolus to the liver. Busch observed that retrograde movement of the blood from the right heart takes place in the vena cava, and in this way hepatic emboli may occur.

Number: Embolic or pyemic abscesses are usually multiple, rarely single.

Seat: For reasons previously given, the right lobe of the liver is more frequently involved than the left, yet in embolic abscess any part of the organ may be the seat of circumscribed suppuration. Unlike the amebic abscess, embolic abscesses are frequently near the surface of the liver.

Size: They vary in size from a minute suppurative area to extensive destruction of an entire lobe. Large abscesses are due to the coalescence of many smaller ones.

The pus of an embolic abscess is always septic. Leucocytic reaction is marked. The surrounding liver-tissue shows congestion and inflammation.

Abscess from gall-stone presents no features peculiar to itself, except the complication of cholecystitis and cholangeitis. It is asserted that the suppurative process begins at the origin of the bile-ducts, but as we have shown in the etiology, the infection attendant upon gall-stones, round worms, etc., takes place through the cystic veins, tributaries of the portal. Abscesses originating from these mechanical causes are, therefore, embolic in character, and present the same general pathological picture.

4. **Tubercular Abscess:** Authorities describe a very rare form of tubercular abscess of the liver, in which the whole organ becomes honey-combed with cavities varying in size from a pea to a walnut. The pus is thick, caseous, bile-stained, and the walls of these abscesses contain the bacilli. It is attributed to tuberculous infection of the bile-ducts. MacKenzie describes a case of multiple tubercular abscess of the liver, in which the right lobe was very much shrunken, but in it there was a globular swelling about the size of an orange on the lower border in the vicinity of the gall-bladder. The remaining portion of the liver was studded by a number of yellow prominences. These small abscesses were scattered throughout the substance of the organ. The contents of the abscess-cavities was soft pultaceous matter. A capsule of connective tissue becoming calcareous surrounded the abscess. Sections of the wall of the abscess showed abundance of bacilli tuberculosis. Efforts to detect the actinomyces were negative.

Hydatid cysts of the liver not infrequently undergo suppuration, the features of which are considered under their proper head.

Concomitant Pathology.—It is generally stated that in tropical or amebic abscess the spleen is not enlarged. In studying many of the cases reported with this point in view, we find that the records do not accord with text-book authority; and where this organ has been mentioned at all, it is almost a universal statement that it is enlarged. In pyemic or embolic abscess the spleen is often the seat of suppuration; if not, it is generally found greatly congested and enlarged. In ordinary traumatic

abscess without pronounced septicemia there is no alteration in the spleen.

In pyemic abscess the focal suppuration sends out infective agents which may find lodgment in any organ or portion of the body, giving rise to a multiplicity of abscesses of widely different locations, the liver being only one of many organs infected.

Leucocytosis is present in all cases, with the possible exception of very small, chronic abscesses, so that when hepatic abscess is suspected examination of the blood should never be omitted, since the presence of leucocytosis is always strong evidence of suppuration, and may become a valuable aid in the diagnosis.

TERMINATIONS.—The patient surviving, there can be but two terminations of hepatic abscess: (1) caseation, absorption of liquid contents, and perhaps calcification of the solid particles, which becoming encysted may remain indefinitely; the proof of this behavior is the finding of true cicatrices in the hepatic parenchyma; such a result must be regarded as quite exceptional.

(2) Rupture of the abscess with discharge of its contents is a common occurrence, yet its actual frequency is difficult to determine. Of Dabney's 108 cases rupture occurred in 18. As we have pointed out, solitary abscesses are, as a rule, near the convex surface of the liver. Their seat explains the great frequency of upward perforation through the diaphragm. Chvostek found that of 327 cases 178 spontaneously perforated the diaphragm and discharged through the lung. Waring records 42 cases rupturing in this direction. In these high abscesses there may be simple perforation into the pleural sac, forming an empyema. Several instances are recorded of perforation into the pericardium; Jacobsen recently reports such an one. Should an abscess upon the convexity of the liver spontaneously burst without diaphragmatic perforation, the result would be the formation of a true subphrenic abscess.

Abscesses pointing upon the under surface of the liver may rupture into the stomach or intestines; 26 per cent. of Chvostek's cases chose this direction. Perforation into the general peritoneal cavity is third in point of frequency; about 15 per cent. of cases may take this course. The discharge of the abscess through the external abdominal parietes is still more unusual

and not so favorable a termination; Chvostek records 9 instances. Cases are reported of the discharge of the abscess through the gall-bladder or common duct. A few cases are reported of perforation into the pelvis of the right kidney. Waring cites three instances of rupture of the abscess into the hepatic vein. Flexner records two cases of amebic abscess of the liver opening into the vena cava, the patient in one dying of hemorrhage eleven days after operation for evacuation of the abscess; at autopsy two perforations into the vena cava were found.

It must not be forgotten that hepatic abscess may burrow in the parietes either upward toward the axillary space, backward into the lumbar region, or downward into the inguinal. Hall, quoted by Schmidt, saw a case in which the pus reached the scrotum.

The discharge of a large abscess that is associated with multiple smaller ones in no way abates the symptoms, nor does the spontaneous discharge necessarily indicate that the abscess is solitary.

General Symptoms.—There are cases in which not a single symptom of abscess exists during life (Frerichs). Fortunately the clinical diagnosis of abscess of the liver is not usually shrouded in such obscurity. The record generally runs as follows: The patient is a man, approaching middle life, somewhat addicted to alcohol and high living, with, perhaps, a former residence in a hot climate, a history of bowel trouble—not necessarily a severe attack of dysentery; positive statements as to bowel disorder are frequently wanting. For several weeks he has been complaining; he has lost flesh, his appetite has deserted him; he is afflicted with insomnia and with a dry annoying cough, especially at night; he is depressed, melancholic, and complains of pain in the right side or hypochondrium; the pain is described as dull, heavy, aching, increased by change of posture; he prefers to lie upon the left side. His complexion is a pale earthy hue, and there is rarely jaundice; his tongue is coated brown or white. There is usually increase of pulse and rise in temperature, and the temperature record in its variability is significant. Night sweating is not unusual.

Physical examination reveals enlargement of the liver, chiefly in the upward direction. Tenderness is usually pronounced over

the lower costal regions. The intercostal spaces are widened, and the superficial abdominal veins enlarged. Fluctuation and localized edema may be found. With such a history and symptoms presented, puncture or exploratory incision will usually reveal a solitary abscess of the liver.

In the study of the diagnosis of abscess of the liver in general, we must embrace the etiology, subjective and objective symptoms. The clinical diagnosis of the varieties of abscess, whether single or multiple, location, probable complications, and amenability to surgical treatment all rest not alone upon the cause, for, as we have shown, amebic abscess may be multiple and septic abscess may be single, but upon the particular objective signs in each individual case. Refined as our diagnostic methods may be, there will always remain a certain number of cases in which exploratory incision is essential for diagnosis.

1. **Amebic Abscess:** In from 50 per cent. to 80 per cent. of amebic abscesses of the liver the history will disclose the previous existence of dysentery. Usually hepatic symptoms will arise in the fourth or fifth week of the intestinal trouble; or months may have intervened and so slight may have been the dysentery that all knowledge of it has escaped memory, and its causal relation is difficult to trace. There are a few cases in which initial bowel disturbance is absent; if such cases reveal at operation or autopsy the presence of amebæ, we must conclude that the infection took place by the alimentary canal; and if the pus is sterile both of amebæ and pyogenic cocci, we shall not admit that it is a primary spontaneous suppuration, but must insist that it is a secondary condition dependent upon an undiscovered source of infection. In any case of lingering illness the history of an attack of dysentery, or milder intestinal disturbance, should suggest the possibility of amebic abscess. Its symptomatic development is slow and insidious; gradually its objective signs are evolved, and a study of these from its initiation through its progressive stages is necessary to comprehend the varied clinical phenomena of hepatic abscess.

Pain: In suppurative hepatitis local pain is practically always present; it is not necessarily a continuous symptom throughout the course of the disease, but it is pronounced at some time. Usually, severe at first, it gradually subsides in intensity, and

again becomes marked from the development of peritoneal adhesions or diaphragmatic fixations. The complications of rupture in any of its directions, pleuritis, peritonitis, etc., excite pain peculiar to the structures becoming involved. The pain of abscess may be distinctly paroxysmal. In a case recently under my observation, the patient at all times complained of a weight and heaviness in his side, but usually in the afternoon when his temperature was highest there would be a distinct exacerbation of suffering, which would spontaneously subside in the course of an hour or two. This observation extended through a period of about three weeks. Patients seldom complain of throbbing pain unless the abscess is attached to or penetrating the parietes. The pain is in the region of the liver over the lower portion of the thorax. If the abscess occupies the under surface of the liver the pain may be referred entirely to the abdomen. In the formative stage of the abscess there is usually pain over the organ itself, and an additional referred or sympathetic pain felt in the right shoulder, but this is not peculiar to abscess. This referred pain is especially marked in those cases of abscess on the convex surface of the liver; it may not be noted in deeply-seated abscesses. It radiates to the neck, upper part of the arm, and is most intense at the spine of the scapula. It is explained by the anatomical connection between the third, fourth and fifth cervical, and the phrenic nerves. Pain is a fairly constant symptom of abscess of the liver. It must not be exaggeratingly described as acute suffering, and is often only a feeling of great heaviness and discomfort.

Temperature: In amebic abscess the temperature is usually intermittent, may be remittent and of higher grade than that attained in ordinary course of the dysentery (Lafleur). Diurnal exacerbations, usually in the afternoon, sometimes preceded by chilly sensations or a distinct rigor, and generally passing off with profuse colliquative sweating, are the phenomena present in amebic abscess, and so frequently ascribed to malaria.

Sweating: As above indicated, sweating usually follows the exacerbations of temperature, but a valuable diagnostic point is that drenching perspiration occurs without preceding rise of temperature, generally during the relaxation of sleep, night or

day. Its occurrence adds much to the patient's anxiety; he attributes his great weakness to the sweating.

Pulse: At first the pulse is but little influenced, remaining 90 or 100; but the daily occurrence of high temperature with increased pain at the time, and the profuse sweating are soon accompanied by the fast and variable pulse of sepsis. In a soldier recently returned from the Philippines, the morning pulse would be under 100; temperature at no time in the twenty-four hours would exceed 102°; yet in the one week in which he was under my observation prior to operation, his pulse at night would vary from 120 to 140. I may say that in this instance it was no doubt influenced by insomnia and great mental depression, which usually possessed him at night.

Insomnia: There is a great wakefulness, not due to the pain or to the fever, but more to the melancholia, the peculiar hypochondriacal condition of the patient. In hot countries this sleeplessness is considered characteristic.

Anorexia: In all cases that I have seen—and it is a statement somewhat general with authors—there is complete loss of appetite. In this connection I would emphasize the observation of Musser that unexplained loss of appetite, nausea or revulsion at the sight of food is highly suggestive of pus-absorption. This author reports a case of abscess of the liver in which there was an entire absence of fever, and improvement in the general condition, yet upon loss of appetite, upward enlargement of the liver, and the history of dysentery, the diagnosis of amebic abscess of the liver was correctly made. Other digestive disturbances, such as occasional vomiting, coated tongue, diarrhea, flatulency, etc., may be observed during the course of the disease. Vomiting is said to be especially conspicuous when the lobus spigelius is involved. If an abscess is upon the under surface of the liver, compressing the bile-ducts or exciting peritonitis, then the gastric and abdominal symptoms are pronounced.

Tussis Hepatica: A dry, distressing cough, without physical signs of pleuritis or pulmonary disease or inflammation, occurring in connection with the above symptoms, suggests involvement of the diaphragm by hepatic abscess. The singultus which is sometimes an annoying symptom may be similarly explained. Those abscesses near the upper surface give rise to thoracic symptoms.

Dyspnea: In large abscesses the diaphragm is forced upward, the lung compressed, and the heart displaced, thus giving rise to mechanical dyspnea.

PHYSICAL SIGNS.—**Inspection:** The disturbance of the function of so important an organ as the liver in suppurative hepatitis produces a picture of a graver disease than the subjective symptoms will usually account for.

The patient occupies a peculiar decubitus. He prefers his head and shoulders elevated, and generally rests more comfortably half-turned to his left side. Change of posture is likely to cause pain. We observe an emaciation which has been in progress oftentimes before the occurrence of any special symptoms. The pronounced anemia, with its peculiar earthy hue of skin and clear sclera, is significant. As a further evidence of blood-disintegration, purpuric spots may appear. Jaundice is rarely present except in abscesses of downward evolution, or in those in which previous hepatitis from cholelithiasis or other causes has existed.

The right hypochondriac and lower thoracic regions are enlarged, intercostal spaces widened, and respiratory movements of that side restricted to the upper portion of the chest. The epigastric veins stand out in distinct blue lines. Not infrequently in the latter stages of the disease there is distention of the abdomen; this may be either tympanitic or ascitic. Edema of the extremities is usually a late symptom.

In abscess the contour of the liver is generally preserved; this is explained by the resisting capsule, the abscess increasing in size at the expense of the parenchyma. Yet we may find a circumscribed visible protuberance elevating the parietes; this is especially true in an abscess of the left lobe. Discoloration and acumination of the parietes indicate adhesion with approaching external rupture.

Palpation: In the subjective symptomatology we have emphasized the constancy of pain. No less important is the hepatic tenderness; it is not definite or fixed in location. If the fingers are pressed deeply under the costal margin, pain is excited on inspiration. Deep pressure over any part of the liver may excite pain. The pain and superficial hyperesthesia in the early stage has been mistaken for intercostal neuralgia.

By palpation we note the stationary thorax, the wide, tense, but not bulging interspaces; in large abscesses, slight displacement to the left of the apex beat of the heart; irregularities in the contour of the liver, especially on the under surface and left lobe. Perihepatic friction may be discerned; and in some instances distinct pulsation, simulating aneurism, has been noticed. In abscess the tumor may be felt in the lumbar, right hypochondriac, epigastric, or even in the left hypochondriac regions. It is always very tender, and occasionally fluctuation may be elicited. It is usually of rapid growth, yet it must not be forgotten that it may be extremely chronic in its duration.

Unfortunately, in only a few cases of abscess of the liver have we the diagnostic assistance of a palpable tumor. Localized edema may be regarded as a fairly certain sign of underlying pus. It is not necessary that adhesions should be present before this edema appears; I observed a case in point in which edema materially assisted in confirming the diagnosis, and yet at operation there were no adhesions. The peritonitis and perihepatitis are indicated by the tenderness and resisting muscles. Seventy years ago Twining observed that at a very early stage of these affections, when hepatic disease is seated in the right lobe, the right rectus abdominis muscle is more tense than the left, and it resists pressure by an involuntary resiliency. This is nothing more than the muscular rigidity upon which we lay so much stress to-day as indicating acute intra-abdominal disorders. It does not exist as an invariable symptom in so chronic an affection as abscess of the liver often is, and would not be present in those cases of upward evolution.

Percussion: Of the objective signs the enlargement of the liver as outlined by percussion is the most important. According to Sachs, this enlargement is first upward, then downward and backward. The right lobe of the liver pushes up the diaphragm so that this part of the organ protrudes more or less into the thoracic space. This is true of abscesses upon the convex surface of the liver. Where we have upward enlargement of the liver, with the semiglobular or curved line of percussion dulness with convexity upward, and the crowding in of the boundaries of lung and liver, we have in these three what Pel regards as pathognomonic and classical signs of abscess of the

right lobe of the liver. Adhesions to the diaphragm and parietes usually prevent sinking of the organ, so that in very large abscesses the liver-margin may not be abnormally low. If we have downward enlargement secondary, so to speak, to the upward enlargement, it points to a very large abscess, and excludes tumors of the liver, whose primary growth is downward.

The enlargement of the spleen, as outlined by palpation and percussion, is of frequent occurrence in amebic abscess. It may develop simultaneously with the hepatic enlargement, but in many cases it is the effect of malaria and has preceded the dysentery.

Auscultation: If seen early, before complicated by pleuritic effusion, friction-sounds may be heard. But little attention has been paid to this diagnostic feature. Lochboehler, who has reviewed the literature with this point especially in view, finds it dwelt upon by only one author. He quotes at length a case reported by Manson, in which a loud bruit of the classical wood-sawing type was heard, synchronous with the systole of the heart, which extended from the upper border of the sixth rib in the nipple line two and a half inches downward and backward to the axillary line. The lower border was distinctly defined. In one point it was high-pitched and had quite a musical character. Lochboehler concludes that these were friction-sounds produced by the adhesions between the liver, peritoneum, and abdominal wall. We may hear the heart-sounds well conducted over the area of dulness.

Hatch calls attention to what he names a hepatic odor in patients suffering from hepatic abscess.

Examination of Urine: Semmola and Gioffredi attach the greatest significance to the daily examination of the urine. At first there is an increase in urea, which is due to the irritation in the hepatic cells. Later, when there is destruction of a considerable portion of the parenchyma, there is hypo-azoturia. "This is a symptom which has always been of especial interest to us, and which we can affirm to be constant, as we have found it in every case of hepatic abscess in our practice" (Semmola and Gioffredi).

Leucocytosis: Of five cases of abscess of the liver in which the blood was examined by Cabot, in all save one there was marked leucocytosis. In chronic amebic abscesses it is often wanting.

Exploratory Puncture: The withdrawal of pus through the aspirating needle is the only positive sign of abscess of the liver. The effort may be unsuccessful: we may fail to strike the abscess-cavity; or the contents may be so viscid that they will not pass through the largest needle. Aspiration is almost universally recommended as a proper procedure to confirm diagnosis. If symptoms and signs are sufficient to warrant aspiration, then it should be undertaken as a step preliminary to incision, which should follow at once if affirmative evidence of abscess is afforded. If localized edema is present, aspiration should be made through this area; if not, we may choose the seventh interspace in the mid-axillary line, or over the center of the area of dulness, or be guided by some point of extreme tenderness.

2. **Multiple Embolic Abscess:** Multiple pyemic abscess of the liver, whether secondary to a general pyemia or consequent upon infection through the portal vein, is essentially the same pathological condition. In both instances from a suppurative focus, wherever located, zoögleæ reach the liver and pyogenesis at once follows. We are now confronted with a disease manifold in its etiology, vague in its symptomatology, but almost universally fatal in its termination. The diagnosis rests largely upon our ability to detect the focus of infection. If an amputation or an osteotomy has been done, if suppurative appendicitis is present, if gastric or intestinal ulcer or puerperal sepsis are facts within the recent history of the patient, then we are alive to the possibilities. But it must appear evident that there are many instances in which the initiating focus has been so insignificant as to have escaped attention, or else so shrouded in obscurity as to have been unrecognized. Then the subsequent symptoms are most perplexing.

If multiple suppurative hepatitis occurs in the course of a general pyemia the symptoms indicative of the metastases in the liver are usually obscured by the general symptoms. The fever still pursues its erratic type, the pulse is the weak, fast pulse characteristic of sepsis; the irregular chills and sweats form part of the familiar picture. An icteroid hue of the skin, or even marked jaundice, gastric disturbances, great emaciation and extreme prostration, delirium, and septic diarrhea all merely denote the general pyemic state. It is only by the physical signs that

we may recognize the hepatic location. Pain, tenderness, uniform enlargement of the liver, evidences of surrounding peritonitis and perihepatitis, are the local signs of a practically hopeless condition. Yet even in general pyemia the abscess may be single or may become so by the coalescence of many, and death is not necessarily always the result.

In those cases of embolic abscess from infection through the portal channels and not attended by general pyemia or metastasis, the disease is not so violent in its onset, nor is it obscured by multiple pathological manifestations. The initial symptoms point directly to the liver, but after suppuration has taken place there, the subsequent course and symptoms do not materially differ from those in the truly pyemic type. The adynamic, typhoid condition of the patient, his rapid emaciation, his occasional chills and fluctuating temperature proclaim the unmistakable septic character of the condition. The greater possibility of the process confining itself to one portion of the liver, and its occasional relief by surgical means, are the only practical differences between the two forms.

Differential Diagnosis.—In reviewing the semeiology of hepatic abscess, we find that it is a disease presenting many phases. The subjective symptoms may alone be conspicuous; and we are confronted with chills, fever, and sweats, which recur with perplexing periodicity. In another instance these symptoms may all be absent; gastric and intestinal symptoms may be especially conspicuous. In others, there may be enlargement of the liver, uniform or tumorous, with marked latency of all symptoms. And in yet another, the disease may simulate intrathoracic disorders. Unless guided by the history and by a careful analytical study of all objective and subjective symptoms, not forgetting the importance of urinary analysis and blood-examination, one may fail to make the diagnosis, and the disease will pursue its course to its fatal termination. Many conditions simulate hepatic abscess. Differentiation must be made from the following:

From **malarial toxemia** the examination of the blood for hemocytozoa, and the quinia-test should determine the diagnosis.

Acute miliary tuberculosis presents at first greater difficulties; but later the detection of the bacillus tuberculosis, and the ab-

sence of any conspicuous hepatic symptoms, reveal the true nature of the infection.

Tertiary Syphilitic Hepatitis: The anemia, progressive emaciation, general debility, digestive disorders, gradual onset, and pains in the hepatic region are symptoms presented in this affection which have led to errors in diagnosis. The absence of decided hectic fever, chills, and sweats, the irregular enlargement, especially downward, the nodular surface of the liver, the extreme hardness, the long duration, and the history and evidence of syphilis, should make the discrimination from hepatic abscess. If need be, the test of antisyphilitic treatment may be employed.

Suppurative Cholangeitis: The intermittent hepatic fever occurring in the course of this disease, and the persistent tenderness in the liver-region, beget a confusing resemblance to hepatic abscess. The differential points are the history of biliary colic, the absence of enlargement of the liver, the transient attacks of jaundice, the exquisite point-tenderness at the tip of the ninth rib, and occasionally a pyriform tumor caused by a distended gall-bladder.

Distended Gall-Bladder: Distention of the gall-bladder consequent upon obstruction to the cystic duct or ductus choledochus with pus, mucus, or bile should offer but little difficulty in differentiation from abscess. The pyriform movable tumor, elastic or fluctuant, the normal size of the liver, the history of biliary colic, and the absence of septic symptoms or focus of infection, incriminate the gall-bladder as the seat of the trouble.

In **pyothorax** the history of pneumonia or pleurisy, the general symptoms of intrathoracic disease, the area of percussion-dulness changing with posture, the upper line of dulness low near the spinal column, rising in the middle near the scapula, and sloping down as it approaches the front, resembling the letter S as described by Garland; is characteristic of pleuritic effusion. Moderate displacement of the apex beat to the left may be found in hepatic abscess, but great displacement rather indicates empyema. In empyema the liver is displaced downward.

Suppurating Hydatid Cyst: This condition cannot be distinguished from abscess of the liver without exploratory operation; and many hydatid cysts are sterile. It is the withdrawal of the

peculiar brownish-red pus, the anchovy-sauce-like material, which is characteristic of disintegration of the liver-cells with the admixture with blood. Since hydatids have a true cyst-wall, the contents show neither liver-detritus nor blood.

Subdiaphragmatic abscess usually contains gas, and when so, may be distinguished from abscess of the liver, first, by the percussion-sound, which shows tympany intervening between the absolute hepatic dulness below and impaired resonance above; second, by auscultation breath-sounds, which are heard below the level of dulness, and descend with inspiration; we also have amphoric respiration and the bell-sound. If the subphrenic abscess does not contain gas, differentiation is exceedingly difficult. Aspiration in pus of hepatic origin might show amebæ and liver-cells; if not, the distinction could not be made.

Amebic abscess of the liver must be distinguished from a large *embolic abscess* due to general pyemia or portal-vein infection. It offers no difficulty in cases of pyemia; the general condition of the patient, the known primary source of infection, and the multiple metastases make it a very clear picture. But in some instances of abscess secondary to an infection through the radicles of the portal vein the cause is obscure. It may be said in a general way that if the patient has not lived in a warm climate, and has not suffered from dysentery, the probability is that the hepatic abscess is septic and not amebic; and it may be further said that, being septic, it is in all probability multiple. It must not be forgotten that the course of an amebic abscess is usually chronic, while that of general pyemia or pylephlebitis is comparatively acute.

Prognosis.—The prognosis of hepatic abscess is generally unfavorable. Multiple abscess from any cause rarely admits of cure. Single abscesses due to gall-stone, suppurating hydatids, flukes, or traumatism, present a favorable prognosis. Of 418 cases of suppurative hepatitis due to various causes, 325 died; the average duration of the cases that recovered was 143 days (Chvostek). In solitary abscesses Ughetti found in 48 cases not operated upon a mortality of 76 per cent.; in 45 subjected to incision and drainage, a mortality of 42 per cent.; in 38 that discharged by the bronchi the mortality was only 14.6 per cent.

In amebic abscess we find the prognosis far more favorable when subjected to operation. In 21 cases operated upon by Smits, there were only 3 deaths. Fontan, who has had the unusual experience of operating upon 40 abscesses of the liver, has obtained 80 per cent. of cures. He says that the average mortality in the tropics is from 37 per cent. to 51 per cent. In the New York Presbyterian Hospital there were admitted during the last twenty years 28 cases of suppurative hepatitis due to various causes; of these 8 were moribund on admission; of 14 subjected to operation 2 recovered; 1 recovered by spontaneous rupture through a bronchus, giving 3 recoveries and 17 deaths in 20 cases. I have treated by operation six cases of large hepatic abscesses, with 3 recoveries and 3 deaths.

TREATMENT.—This, of course, is entirely surgical. Every case of liver infection should be drained as soon as it is diagnosed. For anything like a timely operation, it will often be necessary to aspirate or explore the liver.

While aspiration is recommended and practised by the majority of surgeons, it cannot be done with safety and satisfaction in those cases in which it is most necessary unless the liver has been exposed by incision. This plan of procedure (exposure by incision before aspiration) is undoubtedly best for those cases in which the abscess is so small that there is little increase in the size of the liver. It may be said that when aspiration is necessary for diagnosis, it should only be done after the liver has been exposed by incision. If merely used for confirming a diagnosis, then it may be used before incision is made, but not until every preparation has been made for the operation which is to follow immediately if abscess is proved to be present.

The incision ordinarily employed for exploration is vertical just within the right linea semilunaris, but an incision along the costal margin can often be used with great satisfaction, as it gives better access to the liver. The objection to this incision is that it cannot be closed with the same security against hernia as the other.

The abscess once located can usually be drained most readily by incision through the thoracic wall after the resection of one or more ribs. If it is necessary to go through the pleura in making this incision, and it is found that the costo-phrenic sinus is not shut off by adhesions, the wound should be packed with gauze for a couple

of days before opening the abscess. Before the wound is packed the diaphragm should be incised carefully. If it is found that there are no adhesions protecting the peritoneal cavity, the gauze should be packed directly upon the liver in order to provide at once for the protection of both peritoneum and the pleura. When the parietal incision is made below the costal margin, and it is found that there are no adhesions protecting the peritoneum, the two stage operation may be done if the abscess is large and accessible, otherwise immediate incision should be used after careful packing off with gauze. If the abscess is to be drained at once, the cavity should be partially emptied by aspiration before the liver is incised, and after incision the pus should be mopped out, and should not be allowed to flow over the field of operation. In opening deep abscesses the actual cautery may be used to prevent hemorrhage. The incision in the liver should be as free as circumstances will allow and large double rubber tube drains should be employed. Irrigation should not be used at the time of operation unless the peritoneum is protected by adhesions.

Neither aspiration with the injection of a solution of quinine or other drugs, nor evacuation by trocar and canula with the introduction of a drainage tube through the opening made in this way, can be advised as a substitute for free incision.

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ECHINOCOCCUS OF THE LIVER.

DEFINITION.—A cystic disease of the liver due to a larval form of the tenia echinococcus.

SYNONYM.—Hydatid cyst of the liver.

FREQUENCY.—It is said that one person in every seven in Iceland becomes infected with the parasite. It is very common among the shepherds of Australia. Less than 100 cases have been reported in the United States. Occasional cases occur in every clime. The parasite may find lodgment in any portion of the body. The relative frequency of hydatids of the liver compared with their occurrence all other parts of the body together, according to Davaine, is 166 in 376 cases; Böcker, 27 in 40 cases; Neisser, 451 in 900 cases.

ETIOLOGY.—The dog and animals of the same family were formerly supposed to be the original host and medium of infection of man by the echinococcus. Its frequent occurrence amongst those peoples who are thrown into immediate contact with their dogs, and the comparative exemption of all others, strongly sustain this view. But Richardson, in 1867, and later Madelung, have shown that echinococcus cysts are frequently found in sheep, and that the eating of mutton so infected is a possible means of contagion. However conveyed to man, it is the ovum of the tenia that is swallowed in food or drink, the capsule of which, being acted upon by the intestinal juices, is digested, and the embryo is liberated. The echinococcus in this larval state is globular in shape, and possesses six hooklets, by the aid of which it may bore into the tissues and find lodgment in any portion of the body. But more frequently it enters directly into the radicles of the portal vein, and is carried from the stomach or intestines through the blood-channels directly to the liver. It may also find entrance to the liver through the bile-duct.

Age: It is most usually met with between the ages of twenty and thirty years. Greig Smith says that in comparatively young persons a slowly-growing, fluctuating enlargement of the liver,

unattended by any pronounced symptoms, is probably hydatid. It has been met with in the fetus, obstructing delivery.

Sex: It is far more common in men.

PATHOLOGY.—The resulting changes in the liver are usually due to a single parasite; exceptionally, multiple invasion occurs. Once in the liver the parasite loses its hooklets and enters upon its so-called vesicular or hydatid stage. The vesicle develops from the caudal extremity, and enlarges and surrounds the scolex, which is attached to the internal surface of the membrane and floats freely in the fluid; so that now the parasite consists of an enveloping membrane, the scolex, and its fluid contents. It is slow in its evolution, may be months in attaining a diameter of 20 mm., and years in reaching its largest size. The usual site of the cyst is the convex surface of the right lobe. The cyst-wall is very delicate and elastic, and easily detached from the parenchyma. It consists of a variable number of layers; the external one is a laminated, cuticular membrane or capsule; the inner one is called the granular, parenchymatous, or germinal layer. The fluid is non-albuminous, of sp. gr. 1006 to 1012, contains sodium chloride and traces of succinic acid and sugar. It is clear, colorless, and transparent; but when the integrity of the capsule is destroyed by erosion or suppuration, the contents may be blood- or bile-stained. Microscopical examination usually, though not invariably, will reveal the presence of scolices and hooklets, and almost always, shreds of the laminated cuticula. Boynet, of Marseilles, has extracted a ptomain from it in the form of a long, silky, prismatic crystal, shaped like a fern-leaf. If the cyst is fertile, that is, capable of reproducing other cysts, these may develop endogenously from the inner surface of the enveloping sac, or exogenously from the outer surface. Each of these buds from the germinal layer forms a brood-capsule, in which scolices develop; these are provided with suckers and hooklets. Soon a vesicle develops which surrounds the scolices, forming a daughter cyst. Then in turn from these may develop grand-daughter cysts. These offspring may retain their attachment to the maternal cyst, or float free in the fluid. Gardner suggests the terms 'ektocyst' to mean the thick elastic wall, and 'endocyst' the enclosed parasites. The scolex begins as a slight mound-like elevation, and as the brood-capsule grows the number of scolices

increases, so that in old hydatids there may be many thousand scolices. As Gübler has pointed out, as long as the parasite is alive, and in sterile cysts, that is, in those in which there is no reproduction of teniæ, the fluid is clear and non-albuminous; but after the parasite's death albumin is found, and other changes occur in the fluid.

The origin of multiple hydatids in the liver is not fully explained. Könitzer thinks that they are the result of a single infection furnishing numerous germs. He dislikes the term 'multiple echinococci' when applied equally to cysts occurring in the same organ, and prefers the term 'echinococcus multiplex'; he finds about 12½ per cent. of the cases reported to be of this variety. Thompson exhibited before the Pathological Society of Manchester a beautiful specimen of multiple hydatids; two were long since dead, and were converted into inert masses; two had suppurated; and the fifth was in active growth. Vierordt describes a form of multilocular echinococcus of the liver as 'echinococcus alveolaris.' On section the tumor looks like a honeycomb or fine sponge; it is of firm, callous consistency, and the spaces contain a colloid substance which floats upon water, and which was formerly described as 'alveolar colloid' of the liver; it is not distinctly encapsulated, and between the cysts are areas of healthy liver-tissue.

In hydatid disease of the liver the organ may be involved in part or *in toto*. The liver is usually changed in appearance, and more or less enlarged; tumors weighing twenty pounds have been observed. The peritoneum is thickened, and adhesions take place to adjacent organs. The color of the liver is usually white or yellowish-green. As the result of the irritation produced by the echinococcus a firm fibrous wall is formed around the cyst, consisting of the compressed parenchyma of the organ. The cyst receives its blood-supply from the branches of the portal vein and hepatic artery. As the cyst enlarges the portal vein and bile-ducts become obstructed.

Echinococcus cysts may be spontaneously cured by death of the parasite, absorption of the fluid contents, and calcification of the sac. In case of death of the parasite and infection of the cyst, suppuration occurs. The tumor may acquire attachments to the abdominal wall and the contents be discharged externally, which

is the most favorable termination; or it may perforate the diaphragm and rupture into the pleura or pericardium, setting up a fatal pneumonia, pleuritis, or pericarditis; or it may open into the general peritoneal cavity, exciting a rapidly fatal peritonitis. A more favorable termination is for the suppurating hydatid to acquire intestinal adhesions and to evacuate through a communication with the intestines.

An overdistended cyst may rupture from slight violence. Its contents are aseptic, and the fatal results attending its rupture are due to the ptomainic poisoning, one of the earliest symptoms of which is a violent urticaria. Instances are recorded in which the cyst-wall is eroded and its contents are discharged into the bile-channels, the gall-bladder, hepatic or common duct. Berg records an interesting case of this kind, in which the opalescent bodies found in the stools were recognized as daughter cysts. When communication is established between the gall-duct and the cyst, the bile empties into the echinococcus sac and destroys the parasite. Should a communication be established between the sac and the hepatic vein or vena cava, death from embolism will quickly occur. The cyst may rupture into the urinary passages, and spontaneous cure be thus effected. Of course, these are exceptional instances. In case of rupture of an echinococcus cyst, whether suppurating or not, there are usually preformed adhesions. This localized peritonitis and pressure of the tense sac give rise to prodromal symptoms.

Symptoms.—The symptoms in the early period of a hydatid of the liver may be very obscure, indeed, entirely latent, and may not arise until a large tumor develops, which by pressure or complications develops them. Treves says that, as a rule, there is no pain, fever, wasting, or jaundice in hydatids; the tumor is simply burdensome. A careful study of the history of these cases will show that this is not literally true, for even in the initial or latent period there may be symptoms not diagnostic, yet suggestive of hepatic disorder. These prodromal symptoms are a feeling of fullness or stretching in the hepatic region, occasional "irradiation pain of the right scapula," right-side epistaxis, disgust for fatty articles of diet, and attacks of urticaria. These symptoms suggest the seat but not the character of the dis-

order; and not until we have a cyst of considerable size do special symptoms develop.

The subjective as well as objective symptoms depend upon the size, location, and complications that attend the growth of the tumor. If the cyst is located near the upper surface of the liver, respiratory and cardiac disturbances are conspicuous. The patient suffers from dyspnea, asthmatic difficulties, dry cough, and palpitation. If the tumor is located on the under surface of the liver near the hilum, it will give rise to pressure-symptoms while it is yet of moderate size. Obstructive jaundice, ascites, and digestive disturbances, are the most frequent disorders. In tumors of anterior evolution subjective symptoms are practically wanting. The greatest difficulty is in the diagnosis of those tumors that develop on the upper and posterior surfaces. Perhaps the most valuable fact obtainable from the patient is the long duration of his complaint. The vague and inconstant subjective symptoms force us to rely upon the objective signs for a diagnosis.

PHYSICAL SIGNS.—**Inspection**: The appearance of the patient depends upon the duration of the disease and its attendant complications. The bearer of a hydatid tumor of sufficient dimensions to give rise to symptoms evinces by his emaciation the unbalanced metabolism. If the tumor is of moderate size, growing on the anterior surface of the liver, its appearance in the epigastric or hypochondriac regions as a globular mass, from the size of an egg to as large as a child's head, may be the only outward manifestation of the growth. If deeply situated in close proximity to the large blood-channels and bile-ducts, the patient may be deeply jaundiced or show only slight icterus. The superficial abdominal veins are not infrequently varicose, and in large tumors there may be dropsy of the abdomen and extremities. In tumors of the right lobe of the liver, and in those of an upward evolution, the right thorax is spread outward in wing-form, with tension of the intercostal spaces. Petechial spots are sometimes seen on the surface of the body, and ecchymoses of the mucous membranes may occur.

Palpation: In simple cysts the tumor is found to be globular, smooth, usually elastic, and sometimes very fluctuant. It moves with respiration, and is inseparably connected with the liver. By

combined palpation and percussion hydatid fremitus, due to the impaction of daughter cysts against each other, is elicited in about one-half the cases of large cysts. This vibratory sensation or 'hydatid thrill' of Briançon, when obtained, is considered pathognomonic. In multilocular cysts the tumor is often irregular in outline; fluctuation is not easily detected; the surface may feel hard, even cartilaginous.

Percussion: The tumor is dull, and this dulness is continuous with that of the liver. Occasionally the tumor may be pedunculated, and there will be an area of modified resonance between the tumor and the liver.

Auscultatory Percussion: In 1894, Santini by auscultatory percussion discovered a special and peculiar sound of a sonorous quality, having a low tone and of brief duration, ceasing abruptly. This *booming sound* is considered valuable in the diagnosis of hydatid cysts, and in the differentiation of single from multiple cysts. The sound is uniform in single, and shows variation in multiple cysts.

Aspiration: So high an authority as Ségond says that in hydatid cysts of the liver preliminary aspiration should always be done, as it establishes a diagnosis. Dieulafoy considers it applicable not only as a diagnostic, but as a curative measure in about one-half the cases. Aspiration may prove negative because of the sterile nature of the cyst, or because of the thick colloid character of the contents. If pus is withdrawn, it may lead to the conclusion that there is abscess. The dangers are ptomainic poisoning, inflammation of the peritoneum, and dissemination of the germs in the abdominal cavity. When we remember that the tension of these cysts is very great we can accept fully the statement of O'Conor: "I have never yet introduced into a hydatid cyst through an open wound a trochar, hook or needle without seeing an escape of fluid; this is bad enough in a patent incision, but infinitely worse in a diagnostic puncture or tapping." Toxic symptoms occur very quickly after tapping. With this view of the case, it seems doubtful if aspiration is justifiable, even if the diagnosis is not obvious.

In the course of echinococcus cysts of the liver death of the parasite, infection and suppuration may occur. The symptoms presented by this complication do not differ materially from

those of abscess of the liver. Pain becomes severe and radiating, tenderness is marked, the tumor increases in size; and there are present the general symptoms of sepsis; it excites special symptoms in the direction of its pointing. It usually bursts upward into the thoracic cavity, giving rise to pleurisy or pneumonia of a fatal character. In rarer instances in which communication with a bronchus is direct, expectoration of the cystic contents takes place with resultant recovery. It may open in other directions, as noted in the pathology. Should an echinococcus cyst open into the gall-bladder, common or hepatic duct, marked symptoms of biliary colic with obstructive jaundice will appear. The detection of the daughter cysts in the stools will make positive the diagnosis of the escape of the echinococci. The parasites may reach the intestinal canal from the liver, also, by rupture of the cyst into the stomach, duodenum, or intestines. When rupture into the stomach or duodenum takes place there will be emesis of the characteristic fluid mixed with ingesta or blood. Rupture into the general peritoneal cavity of a non-suppurating cyst usually produces death by shock or ptomainic poisoning, yet instances are recorded of recovery from this accident. Eichhorst calls attention to a highly-characteristic aromatic odor, resembling that of boiled plums, which he believes to be of great value in the diagnosis of impending or actual perforation of the cyst.

DIFFERENTIAL DIAGNOSIS.—

Echinococcus of the Liver.	*Cancer of the Liver.*
Age.—More common from twenty to thirty years.	More common from forty to fifty years.
Duration.—Many years; 12 to 18 months after jaundice appears.	16 to 24 weeks.
Cachexia.—None.	Pronounced.
Growth.—Very slow.	Noticeably progressive.
Tumor.—Smooth, globular, elastic; often fluctuant, generally single.	Hard, irregular, nodular, and generally multiple.
Pain.—None, unless complicated.	Pain a marked feature.
Tenderness.—Absent, unless suppurating.	Generally tender because of surrounding peritonitis.

Echinococcus of the Liver.	*Cancer of the Liver.*
Gall-stones.—No history of calculi.	Attacks of biliary colic common in history.
Metastases.—None.	Often present.
Glandular Involvement.—None.	Present.

Multiple echinococcus cysts, especially of the alveolar variety, giving rise to a hard, inelastic tumor, present the greatest difficulties in diagnosis from cancer. The history of the patient, the duration of the disease, and aspiration are our only means of differentiation. Acute, rapidly-growing cancer is sometimes fluctuating.

Echinococcus of the Liver.	*Hepatic Abscess.*
Sex.—Not unusual in females.	30 to 1 in males.
Duration.—Months or years.	Often a few weeks, rarely more than three months.
History.—None.	Dysentery, gall-stone, appendicitis, pyemia, etc.
Pain.—Not marked.	Generally marked.
Tenderness.—Absent.	Usually present.
Septic Symptoms.—Absent.	Present.
Emaciation.—Not marked.	Pronounced.
Anemia.—Not marked.	Peculiar and marked.

In inflammation and suppuration of hydatid cysts the differentiation from abscess is very difficult, if not impossible. Aspiration may prove negative. At operation the pus from an abscess may show amebæ, and will show liver-cells and detritus, which are not present because of its surrounding capsule in echinococcus cysts.

Echinococcus of the Liver.	*Hydronephrosis.*
History.—Indefinite.	Of renal colic, and of urinary disturbances.
Pain.—Not conspicuous; when present, radiates upward.	Conspicuous, radiates to genitals and thighs.
Tumor.—Of definite or increasing growth.	May vary in size from time to time.
Shape of Tumor.—Smooth and globular.	Oblong, kidney-shaped.

Echinococcus of the Liver.	*Hydronephrosis.*
Movement of Tumor.—Moves with respiration, unless very large.	May move slightly; but if held, will not ascend on inspiration.
Percussion.—Dull over limits of tumor, and continuous with liver. Hydatid fremitus noted in half the cases. Booming tone in auscultatory percussion.	Resonance over inner and anterior border; area of resonance between liver and tumor. Fremitus and booming absent.
Posture. — Knee-chest position may cause tumor to fall away from loin.	Does not.

Pleuritic effusion in the right side may be distinguished from hydatids by the line of dulness, the absence of vocal fremitus, the impaired or absent vesicular breathing, displacement of the heart to the left, fixed thorax, and bulging interspaces.

Distended Gall-Bladder: In any cystic tumor of the abdomen we must not forget the possibility of its being a distended gall-bladder. This should be distinguished from echinococcus cyst by the movable and pendulous character of the tumor, its pyriform shape, the history of gall-stone colic, and probably icterus. The course of the disease is necessarily more acute.

PROGNOSIS.—The injurious effects of echinococcus cysts are due to ptomainic poisoning, to mechanical destruction of the parenchyma of the liver, and to the complications which may arise. And while it is a disease of an exceedingly chronic course, it must nevertheless be considered a grave affection. It has a mortality of about 25 per cent.

SURGICAL INDICATIONS.—The surgical indication is to remove, when possible, the cyst and its contents—the operation devised by Bond, in 1891. It has been proved that the cavity left by the removal of the sac may be closed with sutures without drainage. There is little danger of collection of serum or bile in the cavity if the soft, elastic sac is entirely removed. So complete an operation is not always possible. In suppurating cysts it can never be done.

When it is not expedient to remove the sac, then simple incision after suture of the sac to the parietes is the proper procedure. In all cases protection from peritoneal infection must be afforded by gauze-packing. This is more desirable than the operation in two stages. After exposing the tumor, it is advisable to partially empty the sac by aspiration before attempting to suture or incise it. Cysts of upward evolution must be reached by the transthoracic route, exercising the greatest precautions against pleural infection.

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NON-PARASITIC CYSTS OF THE LIVER.

This is a degenerative process, resulting in the formation of single or multiple encapsulated fluid accumulation in the liver.

The various forms of non-parasitic cysts of the liver are either congenital or originate in the liver-cells, in the bile-channels, or in connection with the blood-vessels. This does not include cystic changes in malignant disease, or that rare condition, tubercular infiltration, leading to the formation of minute vomicæ throughout the liver. Single cysts unconnected with the bile-channels are sometimes found in the liver, and Virchow has shown that this variety is congenital. After an acute illness in which the liver is especially affected, degenerative changes may take place in the liver-cells, giving rise to cysts. A special study of this variety was made by Pye-Smith, and in a contribution to the Transactions of the Pathological Society of London he describes the process of vacuolation. The process commences by the formation of a minute vacuole in one of the liver-cells. This increases in size and pushes aside the protoplasm till, at last, the distended cell bursts. The coalescence of vacuoles ultimately results in the formation of a minute cyst visible to the naked eye, containing brownish-red *débris,* apparently the remains of destroyed hepatic cells. Hale White contributes an excellent study of these cysts, and holds that they are not congenital, since the sections show that the process was still going on at the time of death. The four patients from which the specimens were removed were all adults.

Cysts with epithelial lining are supposed to originate from occlusion of the bile-channels, either by concrements or catarrhal conditions. An abnormal secretion of mucus or bile-products leads to the formation of true retention-cysts in the bile-channels. All authors agree that compression of the bile-ducts may cause dilatation of the biliary radicles. A remarkable instance of a cyst with this genesis is recorded by Müller. The patient was a female 59 years of age. The tumor had existed for ten years.

The symptoms were chiefly those of pressure; latterly, attacks of weakness, which were found to be due to hemorrhage into the sac. The patient recovered after operation of incision and drainage; six pounds of chocolate-colored fluid, composed chiefly of bile and blood, were removed.

There is a certain form of cystic disease of the liver associated with a similar degenerative process in the kidney, in which vacuolation is not present, nor is there obstruction of the biliary channels or apparent disease of the blood-vessels, yet both liver and kidney show multiple cystic degeneration. The nature of this process is not understood; there are two views offered in explanation: the one, that it is initiated in embryonal life; the other, advanced by Juhel-Renol, is that it is a new growth. It is only of pathological interest, as it presents no symptomatic differentiation. The simultaneous existence of cystic degeneration of the liver and kidneys was first drawn attention to by Bristowe.

The symptoms presented in cystic degeneration of the liver are due either to interference of the functions of the organ, or to pressure or complications. The physical signs and symptoms are practically the same as in echinococcus cysts. The non-parasitic nature of the cyst cannot be determined except by an examination of its contents. Single cysts not infrequently become pedunculated and produce much confusion in diagnosis. McDonald reports a case of a cyst with mucoid contents successfully excised from the under surface of the liver.

Cysts of sufficient size to be recognized should be treated by incision and drainage.

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SOLID NEOPLASMS OF THE LIVER.

I. Carcinoma.

Definition.—An atypical epithelial neoplasm of primary or secondary origin.

Frequency.—About 3 per cent. of deaths from all causes are attributed to carcinoma of the liver. It is relatively the most frequent of all neoplasms affecting the liver. Three-fourths of all cases are secondary in origin.

Etiology.—**Age:** Carcinoma of the liver is a disease of middle and advanced life.

Sex: Primary carcinoma of the liver is more frequent in men than in women, while primary carcinoma of the gall-bladder is four times more common in women than in men. The secondary growths appear alike in both sexes.

Primary carcinoma of the liver is so frequently associated with gall-stone that the irritation produced by these concrements is now regarded the most potent factor in the etiology of cancer of the bile-channels and of the liver. The coexistence of cirrhosis with cancer has led many investigators to inquire into their causal relationship. Many instances of this association are recorded. The most carefully studied cases are those of Fussell and Kelly, Waldstein, Finley and Adami. It is difficult to determine the dependence of one upon the other, but we know that cirrhosis of the mammæ appears to bear very definite relations to the development of carcinoma in those organs, and it may act in a similar way in the liver. It is argued with equal force that both cirrhosis and cancer are due to common causes, irritants and intoxicants—mere theory.

Pathology.—(1) **Primary Carcinoma** occurs in three macroscopical forms: (a) as a solitary nodule, usually of large size, generally found in the right lobe; (b) as a diffuse infiltration of the entire organ; (c) as small multiple nodules along the portal vein.

(a) When present as a nodular tumor the growth may attain enormous dimensions, and protrude as an irregular prominence from the surface of the liver. The boundary line between the neoplasm and hepatic tissue is, as a rule, sharply defined, but it may blend inseparably with the liver-tissue. The growth may be affected by hemorrhagic infiltrations, fatty, cystic, or other degenerative changes. Suppuration is not uncommon. The central portion of the tumor usually shows these retrogressions. From the compression of the bile-ducts the liver often shows discoloration, while the growth itself is generally white or yellow in color.

(b) The infiltrating variety, which is but seldom met with, produces general symmetrical enlargement of the liver. Small protuberances may be found at necropsy, but are not usually detected clinically. The slow growth and induration simulate cirrhosis.

(c) In the third or nodular type there are nodular tumors, small in size, along the portal vein or scattered throughout the liver. These tumors are hard and rarely undergo degenerative changes. The intervening parenchymatous tissue of the liver is in a state of cirrhosis.

Microscopic Appearance: Primary carcinoma of the liver does not vary in its histological structure from carcinoma elsewhere. The prevailing type is the cylindrical-celled epithelioma, either tubular or acinous in arrangement.

(2) **Secondary Carcinoma:** It is as a secondary growth that we most frequently meet with carcinoma of the liver. The parent growth is usually in some portion of the alimentary tract, most frequently the pyloric extremity of the stomach, yet it occurs as secondary to cancer of other organs, such as of the breast, uterus, ovaries, testicles, etc. Secondary cancer is a multiple growth producing great enlargement and deformity of the liver by the development of irregular nodules. The nodules are white in color or bile-stained, and visible throughout the liver in every cut surface. They vary from the size of a cherry to enormous tumors. In the examination of the liver Tuffier emphasizes the importance of searching the area of the hilum; if enlarged glands are found, it shows the secondary character of the tumor and that metastasis has occurred, and further interference is then unneces-

sary. But the absence of enlarged glands does not exclude secondary malignant disease, for in cancer of the liver metastasis occurs through the venous channels, and not by the lymphatics, as a rule. In primary cancer the finding of the glands should preclude further incursion. The rapid growth, the tendency to retrograde changes, and the surrounding peritonitis in secondary cancer of the liver may conceal the primary pathology, which is frequently unmarked by characteristic symptoms. The histological structure of the hepatic growth partakes of the character of the parent neoplasm.

In both primary and secondary carcinomata there may be general glandular involvement. The occurrence of ascites may be due to obstructive conditions, to peritoneal irritation, or to growths in other organs. Jaundice is not an essential pathological feature of cancer of the liver; it is due to the accidents of location and size of the neoplasm.

The blood-changes in cancer of the liver are only marked in the advanced stage of the disease, though cachexia is early and pronounced. We find a decrease in red corpuscles and hemoglobin, and presence of leucocytosis—points valuable in differential diagnosis.

Symptoms.—Hepatic symptoms occurring in the course of recognized malignant disease in other portions of the body would naturally suggest the development of secondary cancer of the liver. But those organs which are especially prone to give rise to secondary liver-growths, that is, the stomach, pancreas, and duodenum, may suffer from carcinomatous disease without pronounced symptoms. In every case, therefore, of supposed hepatic tumor a most searching investigation should be instituted to ascertain its possible secondary nature. Due weight must be attached to the habit of the individual, and especially should the history of gall-stone attacks be noted.

The patient is usually of middle or advanced age, though Acland and Dudgeon are able to collect 9 cases of primary carcinoma of the liver in individuals under 20 years of age. Dyspeptic symptoms are generally persistent, with intercurrent nausea and vomiting, the bowels are constipated, appetite impaired, emaciation is marked and progressive. Patients with this disease show a continuous decline in weight; it is, of course, more rapid and marked in the secondary than in the primary growths.

Cachexia: Cachexia, an important symptom in malignant disease of the liver, develops early and advances steadily. It is noted that cachexia progresses rapidly after the occurrence of the hepatic metastasis; it may not have been appreciable when the disease was confined to its original site. The cachexia is probably an auto-infection; innocent tumors of the liver involving greater areas do not produce the same general effects.

Pain: The pain depends somewhat upon the site and size of the growth. Single primary growths in the substance of the liver may cause only a sense of weight and heaviness, while those tumors, either primary or secondary, which encroach upon the capsule cause pain of a sharp, lancinating character. If the tumor is near the hilum, the pain is often paroxysmal, colicky in character, due to biliary obstruction. The associate perihepatitis and visceral adhesions are additional sources of pain. The complaint may be referred to the region of the liver alone, or to the epigastrium, thorax, back, and shoulder.

Pulse and Temperature: Fever may occur, attributable to ptomainic poisoning, to suppurative processes in the tumor, to perihepatitis, or to intercurrent affections. Some elevation of the temperature is the rule in all cases of large tumor or advanced disease. Unless there is marked cholemia the pulse is in accord with the temperature.

PHYSICAL SIGNS.—**Inspection:** The sallow or icteroid hue of the skin, the emaciation, the gloomy, Hippocratic expression, the fixed and bent trunk, and careful gait, are physical signs indicating malignancy in some one of the abdominal viscera. In less than one-half the cases of hepatic cancer there is jaundice, but all patients have a dry, parchment-like, yellow skin. Jaundice does not indicate the stage of the disease, but is determined merely by the accident of the site of the growth; its development materially hastens the fatal termination. The same may be said of ascites and edema of the extremities; they are not essential, but complicating conditions. The significant feature of cancer of the liver upon inspection is the peculiar enlargement of the organ. In massive, single, primary cancer, and in secondary growths of the liver the emaciated condition of the patient, unless the abdomen is greatly distended by ascites, permits the tumorous enlargement of the liver to be seen. The

tumor may appear as a small nodule beneath the costal margins or in the epigastrium; larger growths may fill the abdomen to the pelvis. Tumors near the free margin of the liver by their weight and size elongate their hepatic attachment into a pedicle; such tumors possess great mobility, and may occupy an aberrant position. Petechial spots upon the surface of the body denote blood dyscrasia. Enlarged surface veins may appear, showing the portal obstruction.

Palpation: Primary carcinoma of the liver is usually single, and occupies the right lobe by preference. Quite large tumors may be concealed in the substance of the liver; the tumor may be felt only when it attains to sufficient size to produce an eminence or boss upon the surface of the liver. Single growths occupying the left lobe are felt low in the epigastric or umbilical regions. The tumor is usually hard, but may, on account of cystic or purulent changes, be elastic or even fluctuant. It obeys the movements of respiration, and if pendulous will be quite movable under palpation. Friction-fremitus may be felt. Irregularities upon its surface are due to exogenous growths, omental or intestinal adhesions. Parietal fixation of the tumor may occur, obscuring some of the physical signs. In a minority of instances primary carcinoma occurs as a multiple nodular growth, but, as Rolleston says, a multiplicity of growths is in favor of the secondary character of the tumor. The nodules may be densely hard, others soft, others umbilicated. The surrounding perihepatitis and tension of the capsule causes pain and tenderness under palpation. Outlying secondary growths may be detected; the discovery of them signifies the hopelessness of the condition. A cancerous tumor, either primary or secondary, may undergo sudden and marked change due to hemorrhage or to degenerative processes. In either form of carcinoma the hepatic enlargement is always rapidly progressive.

Percussion: Tumors developed upon the anterior margin and upper surface of the liver are dull upon percussion, and the dulness is continuous with that of the liver. But in those pendulous tumors previously alluded to, there may be an area of modified resonance between the tumor and the liver; deep and careful percussion, however, will develop the continuity of the tumor with the liver. I would emphasize this physical sign, as it has

often led to error, tumors of the liver being mistaken for omental tumors, renal tumors, and floating kidney, because of the supposed intervening area of resonance. Growths on the under surface of the liver, especially those in the neighborhood of the hilum, are resonant upon light percussion. Intestinal adhesions may modify the percussion-note of any hepatic tumor.

Auscultation: Friction-sounds are heard over the hepatic area. In cancers of the left lobe, and in those in the region of the hilum an arterial murmur may be heard all over the organ, due to stenosis of the hepatic artery or to compression of the aorta; this must be borne in mind in differentiation from aneurism.

DIFFERENTIAL DIAGNOSIS.—**Biliary hypertrophic cirrhosis** may be distinguished from cancer of the liver by the age of the patient —usually between twenty and thirty years—by the uniform, smooth, symmetrical enlargement of the liver, by the jaundice, which deepens with the enlargement of the organ, and by the absence of marked anemia or cachexia.

Abscess of the Liver: Cancer of the liver may by infection undergo suppuration. Its differentiation under these circumstances is difficult to make from abscess of the liver. But the history of the patient, the recognized causes of abscess, the conspicuous anemia, the absence of cachexia, and the frequency of jaundice, should make the diagnosis and render aspiration unnecessary.

Syphilis of liver: Multiple gummatous growths, especially when associated with enlargement of the liver, mesenteric nodules, and ascites, present a picture closely resembling malignancy. The age, history, and concomitant phenomena are sufficient to warrant the suspicion of lues in such cases. The test of specific treatment will perhaps be necessary to make the differentiation.

Renal tumors grow from the loin, retain the shape of the kidney, are not influenced materially by respiration, there is an area of resonance between the tumor and the liver, and their relations to the colon may be shown by percussion, aided if necessary by bowel inflation.

Omental tumors usually occupy a lower position in the abdomen, are freely movable, not influenced by respiration, and yield a different note upon percussion.

Cancer of the colon, where it acquires attachment to the liver, presents many objective symptoms in common. The history of constipation alternating with diarrhea, the features of chronic intestinal obstruction, especially the peristaltic patterns, the colicky pains, the absence of jaundice, and the impaired tympanitic note upon percussion should make the differentiation.

Cancer of the stomach: The gastric disorders and hematemesis, the gastrectasia and ischiochymia, and the chemical analysis of stomach contents, are sufficient to point to the seat of the disorder.

PROGNOSIS.—The clinical course of cancer of the liver is progressively downward to a fatal termination. Single primary growths may exist for several months, a year or more, but secondary cancer destroys the patient's life from the twelfth to the twentieth week of the disease.

The operative mortality of 59 cases of all varieties of tumors, collected by Keen, was 15.5 per cent. Several cases of carcinomatous tumor were well two to seven years after removal. Keen expresses the most optimistic views for the future in the surgical management of these hepatic neoplasms.

CONTRAINDICATIONS TO OPERATION.—Secondary carcinoma of the liver is not amenable to surgical intervention. Multiple neoplasms, pronounced cachexia, marked cholemia, and glandular involvement contraindicate all surgical effort.

SURGICAL INDICATIONS.—If the tumor is single, of primary origin, and accessibly located, exploratory laparotomy should be done; and if inspection and palpation of the liver confirm the above facts, *partial hepatectomy* may be undertaken. Out of 35 cases of malignant disease of the liver exposed by operation, Robson has met with only 3 in which it appeared feasible to remove the entire diseased portion; one of these died upon the table; two recovered to die in a few months from a recurrence of cancer. "Every case, save those manifestly beyond relief, should be *explored,*" says Keen, "and the later steps be determined by what is found."

II. ADENOMA.

DEFINITION.—An encapsulated gland-like tumor with a tendency to atypical development.

FREQUENCY.—This is a comparatively infrequent neoplastic growth in the liver, as recognized clinically; it is a not uncommon post-

mortem finding. In 75 operations for tumors of the liver of all varieties, collected by Cushing and Downs, 7 were adenomata. Marckwald has collected 29 cases reported by various observers.

ETIOLOGY.—**Age:** It occurs at any age, the extremes reported being twenty months and sixty-six years.

Sex: It is more frequent in males; Marckwald found that 23 out of 29 cases occurred in men near middle life.

The causes favoring the development of these tumors are very obscure. It is probable that infection, irritants, and intoxicants are common causes in many instances of cirrhosis, adenoma and carcinoma. Coccidial disease of the liver is frequently associated, and is a probable cause of cystadenoma. Schmieden regards the formation of adenoma as a vicarious hypertrophy to replace destroyed liver-cells.

PATHOLOGY.—Adenoma of the liver is always primary. It may exist as a single or multiple growth, which varies greatly in size. Single solid growths may attain to the dimensions of a large orange; they are usually tubular in structure. Not infrequently they develop papillæ from the inner lining, constituting *adenoma papilliferum;* fluid rapidly accumulates in such growths, thus forming a cystadenoma.

In multiple adenomata the liver presents a roughened, nodular surface. It may be infiltrated by circumscribed tumor-masses; these tumors may be minute yellow bodies, or they may attain to the dimensions of the single growth; they are of a grayish-yellow color, sometimes green or of a hemorrhagic tint. The nodules are separated by territories of fibrous cirrhotic parenchyma.

Microscopical examination shows those tumors originating in the substance of the liver to consist of a series of cells resembling liver-cells in an evident state of functional superactivity, showing many nuclei and many nucleoli; or they may arise from the epithelia of the bile-ducts. The icteric coloring of the tumor-cells is explained by the fact that these cells take the place of the liver-cells, and biliary absorption occurs. The cellular proliferation in some instances extends into the radicals of the portal vein, and friable reddish-gray clots, consisting of polyhedral epithelial cells, occupy the lumina of the vessels. The tumor is surrounded by a capsule of dense connective tissue, the remains

of the interstitial tissue of the liver. As a rule, it is a local disease implicating neither lymphatic glands nor distant organs, yet it should be regarded as a malignant tumor.

SYMPTOMS.—The symptoms of adenoma of the liver are largely those of Laennec's cirrhosis, attributable to the compression of the vessels and bile-ducts. It pursues a latent course for a variable length of time. When the growth by its size or multiplicity has destroyed the greater portion of the liver, or has compressed its vessels, symptoms of impeded portal circulation develop. At first there are simply digestive disorders, a distaste for certain articles of food, especially meat, a dull pain in the region of the liver, and meteorism.

PHYSICAL SIGNS.—**Inspection:** Icterus appears early. There is evident marasmus and emaciation. Abnormal enlargement of the subcutaneous veins of the abdomen is seen. Ascites develops rapidly.

Palpation: On palpation we may find a single tumor, hard, circumscribed, somewhat pendulous, moving freely with respiration. In multiple tumors the liver is enlarged, irregular in its feel, and nodose. John B. Roberts records a striking case of multiple cystic adenomata, the mass occupying the greater part of the hypochondriac and lumbar regions. It was irregular in outline and presented near its center a marked elevation with the characteristics, under palpation, of a cyst. This was a case of multiple cystic adenoma, eight or ten cysts being evacuated at operation. An interesting feature of this case is that one of the cysts contained gas.

Percussion: These tumors are dull upon percussion, and in those that project beyond the liver's margin there may be a deceptive area of modified resonance.

DIFFERENTIAL DIAGNOSIS.—The differentiation of adenoma from carcinoma and cirrhosis must be made.

PROGNOSIS.—The duration of this disease cannot be definitely determined. It undoubtedly exists in many instances without giving rise to symptoms. With the development of biliary and portal obstruction the progress of the case becomes very acute. After the development of jaundice and ascites patients rarely live longer than three months.

CONTRAINDICATIONS TO OPERATION.—Multiple adenomata are not amenable to treatment. After the occurrence of ascites and jaundice from single growths the indications are against interference.

SURGICAL INDICATIONS.—If there is only one tumor, and the constitutional condition of the patient permits, exploratory laparotomy should be done; and if the tumor is accessible, it should be removed by *partial hepatectomy*. The operative mortality is not greater than 15 per cent., and the recoveries, from present statistics, are permanent.

III. SARCOMA.

DEFINITION.—A malignant tumor of embryonic connective-tissue origin; it may be either primary or secondary.

FREQUENCY.—Secondary sarcoma of the liver is comparatively frequent. Primary sarcoma is rarer even than primary carcinoma. Leith has been able to collect 25 cases.

ETIOLOGY.—**Age**: It occurs usually in persons under forty years of age; cases have been recorded in very young children. The average age in melanotic growths is forty-eight and a half years.

Sex: It shows no predilection for sex. Melanotic sarcoma, according to Rolleston, is more frequent in men than in women, in proportion of 2½ to 1.

Nothing is known of its pathogenesis.

PATHOLOGY.—As a primary growth it occurs in its usual histological varieties, chiefly the round- and spindle-celled. Secondary growths assume the type of the parent tumor. Melanotic sarcoma occurs as secondary to melanotic tumors of the eye or skin. Primary sarcoma is usually single, and may attain to enormous size. In Boldt's case the liver weighed nineteen pounds and six ounces. Secondary sarcoma is usually multiple. Melanotic sarcoma appears as large black nodules, circular in outline, and separated from each other by areas of condensed white tissue; it is sometimes diffuse. In both primary and secondary growths lymphatic involvement is found. In secondary sarcoma the kidneys and other organs not infrequently participate in the metastasis.

SYMPTOMS.—The early symptoms of sarcoma do not materially vary from those of carcinoma; the digestive disorders, with marked emaciation and local pain, are common to both. The first symptom noticed in Kahlden's case was that the feces were black and the urine dark; this appeared before there was any manifest tumor. Pyrexia is not infrequently present.

PHYSICAL SIGNS.—**Inspection:** We find an emaciated patient with icterus of varying shades; and if the disease has existed several weeks there may be ascites or anasarca.

Palpation: Sarcoma, as a rule, produces very definite enlargement of the liver; the whole organ may be involved in a nodular growth, or the tumor may be comparatively smooth, globular, and attain to enormous dimensions. Mesenteric glands are usually felt to be enlarged, and the liver is adherent to parietes and intestines. Cystic changes or hemorrhages into the tumor may impart to it a distinct sense of fluctuation. It is interesting to note how closely Bramwell's case of enormous primary sarcoma simulated hepatic abscess; aspiration obtained anchovy-sauce-like fluid, which was due to hemorrhage and suppuration. Too, there is always marked tenderness from the perihepatitis, and when the tumor has become adherent to the abdominal wall, it may deceptively simulate pointing.

Percussion: This physical sign applied defines the limits of the tumor and its hepatic relation.

Urinary Analysis: The only diagnostic significance obtainable by means of the urine is the detection of melanin.

DIFFERENTIAL DIAGNOSIS.—**Hepatic Abscess:** Primary sarcoma may be confounded with hepatic abscess. The rapid development, the emaciation, the digestive disturbances, the temperature, the sometimes chronic course, the physical signs, may all be confusing. But the history of the patient, the ascertainable cause, dysentery or infection, his age, usually in middle life, the rarity of abscess in women, hectic fever, profuse sweating, and leucocytosis mark the difference.

It is practically impossible to differentiate sarcoma from carcinoma, though we may be led to a correct conclusion by the age of the patient, the rapidity of the growth, and the local symptoms of greater activity in sarcoma.

PROGNOSIS.—The life history of sarcoma of the liver is usually about four months.

CONTRAINDICATIONS TO OPERATION.—The great vascularity of these growths, and the certainty of recurrence, preclude operation.

IV. ANGIOMA.

DEFINITION.—A tumor composed of the abnormal formation of blood-vessels in thin trabeculæ of connective tissue.

FREQUENCY.—These tumors are of but little clinical significance. Cushing and Down's table of 75 cases of neoplasms of the liver subjected to surgical operation shows that in 4 of the operations angioma was found; none were correctly diagnosed.

ETIOLOGY.—**Age:** Like tumors of this family, they are found in comparatively young persons.

Sex: No predilection for either sex is shown.

PATHOLOGY.—The cavernous nævus is the variety of angioma met with in the liver. They are usually of small size, rarely larger than the fist. They are composed of a net-work of connective tissue, in the cavities of which blood circulates freely.

SYMPTOMS.—In Hanks's case the symptoms were those of carcinoma. Exploratory operation revealed the true nature of the tumor, and no effort was made to remove it. The digestive symptoms subsided, and the patient was apparently restored to good health, the tumor markedly decreasing in size. Hanks refers to another case in which there was similar behavior. As only one case has been correctly diagnosed, the symptomatology must be very vague. Smith does not regard them as possessing any clinical significance; they rarely attain to sufficient size to give rise to pressure or irritation. Aspiration may show fluid blood.

PROGNOSIS.—When of small size they may exist throughout life without giving rise to special disturbance. One instance, at least, is reported in which a diagnosticated angioma was spontaneously cured.

SURGICAL INDICATIONS.—Keen reports the successful removal of an angioma treated by extraperitoneal constriction. The size and accessibility of the tumor should influence the surgeon in determining on an effort for removal. The extraperitoneal treatment of the pedicle would, perhaps, be the safest technic.

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MOVABLE LIVER.

DEFINITION.—In this condition we understand that the organ is displaced, usually downward, is movable, and permits of partial or complete reposition.

SYNONYMS.—Hepatoptosis, Wandering Liver, Floating Liver, Ectopia Hepatis.

FREQUENCY.—The occurrence of hepatic displacement, sufficiently marked to give rise to symptoms, appears as a comparatively infrequent event. In Ziemssen's Hand-Book of Special Pathology, 1887, Thierfelder was able to collect only 9 cases. Graham, in 1895, tabulated all the cases of movable liver found in literature, and there were less than 70 recorded. It is remarkable how one may find things when one searches diligently for them. From the material obtainable in his private practice, in five months Einhorn found 30 cases of floating liver. Expressed in figures, he finds that 3.7 per cent. of all persons suffering from digestive disorders have movable liver. It is consoling to note that in grouping his cases he makes five varieties—the first, cases unaccompanied by symptoms. Those cases of slight downward displacement in which tension or pressure symptoms are absent, and in which there is no abdominal asymmetry, have no clinical significance, and should not burden literature.

ETIOLOGY.—Simple displacement is to be distinguished from movable conditions of the organ. The liver may be *dislocated* and fixed in its abnormal position by many causes; it may be forced downward by increase in the volume of the contents of the thorax, as from emphysema, pleural effusions, cardiac hypertrophy, etc.; it may be forced upward by increased intra-abdominal pressure; Leube describes a case due to ascites; in vertebral caries we find the liver forced out of position. But in all these instances the liver is fixed in its abnormal position as long as the displacing force is acting, and it retains its anatomical connection with the diaphragm.

A **movable** liver changes its position with the posture of the patient, and parts with its relation to the diaphragm. The vari-

ous changes which take place in the ligaments of the liver permit its prolapse. Displacement to the left side can only be to a very limited degree, owing to the connection with the vena cava. In Faure's case, however, this structure was so loose and elongated that it moved from its position on the right side of the spinal column with movement of the liver.

Various theories are advanced to explain the mobility of the liver. Its etiology was first supposed to be the enlargement; Meissner held that the liver-ligaments became relaxed during pregnancy; many congenital and acquired defects in the suspensory apparatus have been described. Landau considers that there are two factors responsible for abnormal hepatic mobility: (1) laceration or stretching of the ligaments, which may occur suddenly, as from a fall; (2) the enlargement of the capacity of the abdomen.

We note that movable liver occurs more frequently in females, but it is also found in males, hence to sex alone cannot be attributed the causation. Injuries, tight-lacing, and tedious labors, while factors in its production, play only a minor part, else the condition would be far more frequent. It is especially common in multiparæ, yet repeated child-birth alone is not sufficient cause. Looking farther, we find that it is a pathological phenomenon most frequently seen in persons with pendulous bellies. Glénard ascribes it to a general splanchnoptosis. Theodore Schott emphasizes what I regard as an essential link in the etiology, and that is emaciation leading to an absorption of the accumulated fat of neighboring organs, muscles, etc. Therefore, in a multipara with a pendulous abdomen and progressive emaciation we find a most suitable subject for hepatoptosis. Trauma may be an exciting cause.

Pathology.—It follows from the pathogenesis above given that in movable liver there is necessarily elongation of the falciform and coronary ligaments. The prolapse of the organ, with traction on and torsion of its vessels, produces venous engorgement. The enlargement of the liver in these cases, then, is an effect rather than a cause of the displacement. The colon, duodenum, or stomach may occupy the position of the displaced liver. In the condition of great abdominal relaxation other viscera, stomach, intestines, kidneys, and uterus, are displaced. Hemorrhoids,

and varicosities of the veins of the extremities, are other phenomena. Gall-stones in two instances have been found as a concomitant pathological condition.

SYMPTOMS.—The conditions giving rise to movable liver have symptoms of their own, which constitute the history of the predisposing causes of the displacement. The subjective symptoms attributable to the wandering organ are the outcome of several different elements. The mere movement of so large a body within the abdominal cavity gives rise to reflex nervous phenomena; this is observed in wandering spleen, movable kidney, and pedunculated tumors. The circulatory disturbances consequent upon the changed relation of the vessels to the liver in its several positions affect not only the organ itself, but derange the equilibrium of the abdominal visceral circulation. The changed relation of the viscera, especially distorting the biliary channels, impedes the flow of bile and gives rise to symptoms indicative of biliary stasis. But it is to pressure and traction chiefly that the symptoms of movable liver are due. The prolapsed organ through its elongated ligaments makes traction upon the diaphragm, causing respiratory distress; Einhorn calls attention to this class of cases. The pressure upon the intestines excites digestive disorders, nausea, vomiting, flatulency, constipation or diarrhea. The changes in circulation may cause attacks of syncope.

Areilza observed the following symptoms in a boy twenty years old: Every two or three months he would suffer a severe attack of pain, chilly sensations followed by vomiting and loss of consciousness; he would be extremely prostrated for several days; there was no fever or jaundice; the abdominal tumor was diagnosed as a floating liver, and all symptoms were relieved by hepatopexy.

Usually the patient complains of pain in the side in the region of the liver, in the chest, shoulder, or lumbar region. The pain is dull and dragging in character, not infrequently it is paroxysmal. In Allingham's case the attacks of pain and the urinary disturbances led to the erroneous diagnosis by the hospital staff of movable kidney. I find two instances of the association of gall-stones with movable liver; in both cases jaundice was present. Contrary to what we would expect, icterus is not a con-

stant symptom; it was observed once in Thierfelder's collected cases. The distress occasioned by a movable liver is variable; in some it may be very great, in others only slight, but is present to some extent in all. The subjective symptoms are of but little diagnostic value.

PHYSICAL SIGNS.—**Inspection:** The abdomen presents a peculiar appearance: the epigastrium is usually sunken; the right thorax has lost its convexity and is flattened, and if the patient is examined in standing position, the pendulous belly overhangs the pubes, and the lower half of the abdomen is markedly protuberant. Enlarged veins, inguinal hernia, and varicocele are not infrequently observed. Glénard calls attention to a fold of skin that conceals the lower part of the umbilicus, due to traction upon the suspensory ligament. The abdominal deformity depends upon the degree of the hepatic displacement.

Palpation: By palpation we recognize the contour of the liver, its sharp, free border, its incisure, and sometimes its convex surface and even its entire left lobe may be palpated. The organ is usually hard from engorgement, moves freely in an upward direction, more or less so to the right, but cannot be further displaced to the left or downward. A moderately-displaced organ obeys the movements of respiration; but in cases in which the displacement is great, if the patient is in dorsal position, the ligaments are relaxed sufficiently for the slack not to be taken up by the movements of the diaphragm. From gall-stone impaction or constriction of the duct we may have distention of the gall-bladder, yielding under palpation a pyriform, elastic tumor in close relation with the free edge. The relaxed and thin abdominal walls permit of easy examination of other organs. Allingham rejected the diagnosis of renal tumor, which had been previously made, because he was able to outline distinctly the kidney under palpation. Palpation, like percussion, must be made in both the erect and recumbent postures.

Percussion: It is upon this sign that the diagnosis of movable liver largely rests. Assuming that the upper limit of normal hepatic dulness is the fifth intercostal space in the right mammary line, we find under percussion that this is displaced downward to the eighth rib, or even to the margin of the ribs, and a hollow tympanitic note is substituted. The dulness extends

over the entire surface of the liver, and its abdominal limits depend upon the extent of liver-enlargement and degree of prolapse. It may reach to the pelvic brim. Under light auscultatory-percussion we may accurately outline the area of hepatic dulness. Manipulation of the liver or change of posture will change the border-lines of dulness.

DIFFERENTIAL DIAGNOSIS.—Movable liver has been mistaken for enlarged and movable kidney, for subdiaphragmatic abscess, for hydatid cyst, and for omental tumor. Edebohls has seen two cases that simulated fibroid tumor of the uterus. A careful application of the methods of physical examination should eliminate such errors. Difficulties will be presented by those cases in which the mobility is complicated by some associate pathological condition, as cancer, gall-stone, or abscess.

It is of the utmost importance after recognizing a displaced liver to consider and eliminate those thoracic and abdominal conditions which cause displacement and fixation, an essentially different condition from that with which we are dealing.

PROGNOSIS.—Patients with extreme displacements succumb to marasmus, the result of prolonged functional disturbances, or to intercurrent diseases to which they are markedly predisposed.

SURGICAL INDICATIONS.—Those cases not made comfortable by a well fitting bandage and that give rise to distressing symptoms may find relief in the operation of *hepatopexy*. Treves regards this for selected cases a perfectly logical procedure. Terrier and Auvray record that in 11 cases in which surgical treatment has been instituted for movable liver, there have been 8 successes, 1 death, and 2 cases yet to be heard from. In none of the cases could the liver be restored to exactly normal position, and from this they conclude that its good results are due to anchoring rather than to reposition.

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CIRRHOSIS OF THE LIVER.

Attempts have been made to relieve abdominal ascites, due to hepatic cirrhosis, by surgical means. The end sought has been to establish new channels by which portal blood may reach the systemic veins without passing through the cirrhotic liver. This subject has been exhaustively studied by Frazier, and his excellent contribution to the American Journal of Medical Sciences, Vol. CXX, 1900, reviews the history of the operation, sets forth the indications and contraindications, and predicts for it a future in properly selected cases. Weir (New York Medical Journal, Feb. 4, 1899), after some clinical experience, is convinced of the value of the operation.

Frazier thus clearly sets forth the indications and contraindications for the operation: "It should be borne in mind that the operation is not indicated in every case of hepatic cirrhosis with ascites; the operation is absolutely dependent for its success upon the retained function of the liver-cells. . . . Cases in which internal medication and paracentesis fail to afford relief, or, in other words, utterly hopeless cases," are the ones selected for operation unless there is some obvious contraindication. Quoting further the same author: "The cases are so hopeless, the technic so simple, the outlook so promising that the prospects of this mode of treatment becoming an established one seem bright." He collects 14 cases, including one of his own; excluding those cases of failure for which the operation should not be held accountable, of which there are 5, we find that 6 of the remaining 9 were well and free from ascites at periods of three, four, six, twenty-four, twenty-four, and twenty-six months; one improved; one not improved; and the ninth case the author does not account for.

Greenough (American Journal of Medical Sciences, December, 1902) has collected 122 cases in which Talma's operation was done. 105 of these he considers to be due to uncomplicated liver cirrhosis. 31 of these cases died within thirty days after

operation; 60 obtained no benefit; 44 were improved or slightly improved, and 9 of the cases were improved in health two years after operation.

Sears and Lord (Boston Med. and Surg. Jour., Sept. 11, 1902) from careful studies conclude that there is no evidence found to show that the disease is not always progressive. They admit that surgical treatment may be indicated in a very small proportion of cases, "but conclusive evidence is still lacking to show that the ascites in the cases successfully operated upon was due to hepatic cirrhosis and not to coincident conditions." We therefore conclude that the surgical treatment of hepatic cirrhosis is a questionable procedure.

GALL-STONE.

DEFINITION.—A single or multiple lithoid formation in the gall-bladder or bile-ducts.

SYNONYMS.—Biliary Calculus, Cholelithiasis.

FREQUENCY.—A statistical estimate of the frequency of any disease based upon necropsies is, in some measure, misleading; this is especially true of gall-stone. The concrement may have passed from the body; furthermore, unless the autopsy is conducted with this special intent, it may be overlooked. It is, therefore, probable that Schroeder's finding of gall-stone in 12.5 per cent. of all autopsies is none too high. This statement closely corresponds with Naunyn's, 10 per cent. to 12 per cent. Brockbank, in 1,300 autopsies, found gall-stone in 7.4 per cent. Its relative frequency to other surgical affections of the bile-passages is well shown by Kehr, who records 360 operations on the gall-bladder and bile-ducts, of which 307 were for gall-stone.

ETIOLOGY.—**Age**: The formation of biliary calculus is too generally regarded as incident only to middle and advanced age. Lamentable errors occur from a failure to appreciate its possible existence at all periods of life. In 9,000 autopsies Naunyn found gall-stone in 1 in every 30 under thirty years of age; 1 in every 6 after sixty years. Bollinger, whose figures closely correspond, found gall-stone in 2.7 per cent. between fifteen and thirty years, 5.9 per cent. between thirty and sixty, and 15.2 per cent. above sixty years of age. Still collects 20 cases in children, 10 of these in infants; Thomson is able to record 6 cases in the new-born. Holt in his work on "Diseases of Infancy and Childhood," devotes only a few lines to the consideration of biliary calculus, while Rotch in his latest edition mentions it as a clinical curiosity.

Sex: Females are especially predisposed to the development of cholelithiasis. Schroeder, in his autopsies at Strassburg, found gall-stone in 20.6 per cent. of females, and in only 4.4 per cent. of males. Two-thirds of the cases occur in women (Sem-

mola and Gioffredi). The relative frequency is usually placed at 4 females to 1 male.

Everything that tends to disturb the secretion of the bile, to alter its composition, to impede its flow through the biliary ducts, or to favor its stagnation in the gall-bladder, may be regarded as a predisposing cause to cholelithiasis. Such was the teaching of that master-clinician, Trousseau, in emphasizing the views of Morgagni. The apathy of age, arteriosclerosis, and retarded metabolism explain the frequency of gall-stone in advanced life. The conditions incident to parturition, the enforced invalidism consequent upon unrepaired genital lesions, the habitual constipation and relaxed abdominal parietes were considered sufficient reasons for its greater frequency in females. The effect of tight-lacing is to establish an artificial fissure; it leads to the formation of a "corset-lobe" which carries the gall-bladder with it, changing the relation of the bladder to the cystic duct, producing a constriction at their junction; stagnation of bile in the bladder follows, and this is one of the requisite steps in the formation of gall-stone. Bile-stasis may be due to the general displacement of enteroptosis, or to pressure from a movable kidney. Obesity, sedentary habits, and confinement, conditions in which there is surcharged hepatic circulation, are certainly proximate causes. Krauss, with special reference to this point, finds gall-stone exceedingly common among teachers, clergymen, and obese officials; more than 50 per cent. of 472 cases were in brain-workers. That it is not attributable to active brain-work, but to physical repose and confinement, is clearly proved by the frequency with which gall-stone is found in the insane; the combined statistics of Beadles, Gorstell, and Snell, reveal that in 21.22 per cent. of all autopsies upon the insane gall-stone is present. According to Brockbank, heart-lesions seem to double the risk to gall-stone; of the subjects with mitral stenosis 21.8 per cent. had biliary calculus. The passive congestion of the liver and the enforced inactivity of these patients favor bile-stasis, a predisposing cause to gall-stone.

The occurrence of gall-stone in all classes of people, and in all countries disposes of diet and climate as elements in its causation. Some effort has been made to ascribe the lithiasis to insufficient water, or to imbibition of water that contains an abund-

ance of lime; to say that the scanty use of water, or its substitution with light acidulated wine will lead to the inspissation of bile is a mere assumption; to contend that the bases of alkaline waters appear unchanged in the bile is contrary to our chemical knowledge of the constituents of that fluid, and does not provide for functional metabolism. The changed secretion of the liver, the result of diathesis or dyscrasia, the supposed inherited tendency to gall-stone, and its relation to gout and arthritis, are suggested etiological factors which attribute the formation of gall-stones to constitutional causes. This primitive conception of the pathogenesis of calculi is still held by Thudichum, and is designated by Bouchard as the "expression of defective metabolism." The formation of a biliary concrement is a process of precipitation. There must be such change in the composition of the bile as will throw some of its constituents out of solution. Trousseau, Murchison, Thudichum, and Bouchard consider this derangement general in its nature and not dependent upon local causes.

It would appear that the chemical analysis of the gall-stone should guide us somewhat in determining its origin. Cholesterin, the principal constituent of biliary calculus, is but sparingly found in the blood itself, and in the liver not at all except under abnormal conditions; yet, being a constant constituent of the bile, its presence can only be accounted for as a product of the epithelial cells of the biliary ducts and gall-bladder. Under certain conditions of stimulation, cholesterin is elaborated in excess of the capacity of the biliary-acid salts, the glycocholate and taurocholate of sodium, to dissolve it, this excess then being precipitated. The other chief ingredient of gall-stone is bile-pigment, the substance known as bilirubin, which is soluble in an alkaline, but not in an acid medium; stasis, catarrh, and a change in the reaction of the bile lead to its precipitation. Lime, though a normal ingredient of the bile in a very small amount, is derived chiefly from the mucous glands of the gall-bladder; its presence in excess is, therefore, due to some perverted action of these glands. Serum-albumin, the product of the epithelial inflammation, is also present, and it has been shown that this, too, favors the precipitation of the bilirubin-calcium from the bile.

The whole question as to the constitutional or local origin of gall-stone was much simplified by Naunyn's experiments. He first proved that the amount of cholesterin and lime in the bile was constant and unchanging, without any relation whatever to the food ingested, and that while the habits of life tend to the development of calculus, this tendency is not due to the amount of cholesterin or lime in the bile. He maintained that the condition which favored the precipitation of the solid constituents was to be found in the gall-bladder and bile-channels, thus upholding the local against the constitutional origin of biliary calculus. To establish successfully the local or inflammatory theory of the formation of gall-stones, it is essential to show that inflammation of the mucous membrane of the biliary passages is the initiating pathological change. The proof is found in the nucleus of the gall-stone itself, which is a pearly body, and consists of epithelial cells in a state of fatty or hyaline degeneration. Thudichum found the nucleus to consist of pieces of membrane from the gall-ducts. In those exceptional instances in which a fruit-seed (Frerichs), globules of metallic mercury, entozoa, needles, etc., find their way into the gall-bladder or bile-duct, they merely create an atrium for infection, thus giving rise to the catarrhal condition necessary for the chemical alteration in the bile, and they then become the nuclei around which the stones form.

The pathogenesis of gall-stone had evolved from the very vague constitutional causes to the definite and demonstrable condition of local inflammation, and here it rested until 1891, when Naunyn advanced his hypothesis of biliary infection, utilizing, as we are assured by Cushing, the observations of Gilbert, which were made the year previous and which demonstrated the probably infectious origin of cholelithiasis.

The "lithogenous catarrh" of Naunyn now being held responsible for the formation of calculi, investigators were next concerned in discovering the cause of this catarrh. Galippe, as early as 1886, according to Cushing, suggested the microbic origin of lithiasis in general. Welch had demonstrated the bacillus typhosus in the gall-bladder, and had suggested its possible relation to cholelithiasis. The finding of bacteria in certain intrahepatic calculi by Naunyn, the demonstration of the presence of the

bacillus typhosus as a cause of cholecystitis by Gilbert and Girode, Blachstein, Milian, and others, and furthermore, the report of Fournier, who in 100 cases of gall-stones removed at necropsies found living or dead bacteria in the stones in 38 cases, seem sufficient to establish the relation between the presence of bacteria and the formation of the gall-stones. And Hunner collects 10 cases of post-typhoidal cholecystitis associated with gall-stone which have been operated upon and the bacillus typhosus isolated.

The demonstration of the presence of bacteria in the bile, in the wall of the gall-bladder, and in the nuclei of the concrements was sufficient evidence to show that it played a part in the formation of gall-stones, yet the efforts of various experimenters to produce calculi by means of microörganisms introduced into the bladder, unaided by other conditions, have been, for the most part, negative. Mignot was successful in two recent cases. Cushing has exhibited two small calculi of typhoidal origin from the gall-bladder of rabbits. In Cushing's cases at the time of inoculation the biliary apparatus was intentionally maltreated; and he maintains that the mere presence of the microörganisms in the bile may not alone be sufficient to excite the catarrhal process. "Some form of irritation, such as traumatism or some hindrance to the proper evacuation of the gall-bladder is necessary."

There are two views as to the route of entrance of microorganisms into the biliary channels. The one usually accepted is that they ascend through the common duct. Cushing thinks that they enter by the blood through the portal vein. Charcot and Gombault, after ligating the common duct, found that the bile above the ligature became infected. Naunyn's and Netter's experiments were confirmatory in showing the presence of the colon-bacillus. This work established the hemic origin of the infection. Mignot believes that all stones found in the gall-bladder are practically of the same age, and that stone-formation ceases with the death of the microörganism.

So exacting are the enthusiastic advocates of the microörganismal origin of calculi, that we find them grouping or classifying the stones according to the cultures grown from them; thus Gilbert, and Fournier divide biliary calculi into two groups: (1)

those due to the colon bacillus, (2) those due to the typhoid bacillus.

The frequency with which the bacillus typhosus has been found in the nuclei of gall-stones has caused cholelithiasis to be regarded as a sequel of typhoid fever; indeed, McCosh thinks that in his case it was a complication, but the abdominal distress in typhoid fever may cause preëxisting gall-stones to move and give rise to symptoms.

Conclusive as the evidence of the bacterial origin of gall-stone appears, we have noted that Cushing holds that something more than the mere presence of germs is required. Osler raises the question as to whether the stones are formed by the action of germs, or whether the irritation they produce invite microörganisms to the part. Richardson says that in his cases examination for bacterial origin yielded negative results. Hunter, while admitting the potency of bacteria in this special pathological process, maintains that the catarrhal condition of the bile-passages may be due to the excretion of irritating substances by the hepatic cells. In conclusion, it may be said that biliary stagnation, however induced, and a lithogenous catarrh are essential elements for the production of gall-stone, and that the preponderance of evidence would indicate that this catarrh is attributable to infection.

Types of Stones.—Reference must be made and due credit given to Beck for his analytical studies of gall-stones, with special reference to their exposure by means of skiagraphy, yet for many reasons it is desirable to adhere to the classification of Naunyn. He classifies biliary calculi under the following varieties: (1) pure cholesterin, (2) laminated cholesterin, (3) common gall-bladder stones, (4) mixed bilirubin-calcium, (5) pure bilirubin-calcium, (6) rarer forms.

Composition and Physical Characteristics: The smallest stones, which may exist as gall-sand or minute bodies, are usually composed of bilirubin-calcium, homogeneous in structure, shiny, of dark-yellowish appearance, and found in great numbers. Stones of pure cholesterin, on the other hand, are usually very large, frequently single, laminated in structure, presenting radiating striæ, and crystalline in appearance when fresh, though

they become opaque when exposed to the air; they are very light, have a soapy feel, and are whitish in color (Hill).

Common gall-bladder stones, by far the most frequent of all types, present the greatest variety in number, size, shape, and color. In Otto's collection there is a gall-bladder containing 7,802 stones (Ewald). They vary in size from the head of a pin to a cherry. They are not crystalline in structure. Such stones are described as possessing a nucleus, body, and shell; the outer surface is hard and stratified, the inner substance soft, almost pulpy in character. The cholesterin is arranged in layers, between which bilirubin-calcium exists in greater or less quantities. The nucleus consists of altered epithelium and bacteria; the presence of mucin is denied. The hardness and friability of a stone are due to the admixture of lime.

Pure stones of either bilirubin, cholesterin, or lime are rarely found. Specimens from the gall-bladder and ducts usually consist of a mixture of two or all three of these ingredients. Those from the intrahepatic ducts are composed chiefly of bilirubin, and such stones are always small, are gray-black or very black, and have a metallic lustre.

Generally speaking, the smaller the number of stones present the larger the size. In a recent operation I removed from the cystic duct a spherical stone three-fourths of an inch in diameter, and from the gall-bladder 48 stones about the size of a pea, and some 260 others of uniform size and color, not larger than a No. 6 shot. The form of the stone is influenced somewhat by its place of lodgment; within the gall-bladder they may be oval or pyriform in shape, or they may be branched or angular; within the ducts, spherical, cylindrical, or barrel-shaped. If many stones are present, by attrition upon each other facets are formed, giving the stones polyhedral shapes; when multiple, their surfaces are polished by friction. Single stones sometimes present very rough mulberry-surfaces; such types often acquire attachments to the mucous membrane, and are difficult of removal. A non-faceted stone would indicate that there is but one, yet Richardson has removed 10 non-faceted stones from the gall-bladder. They may be opaque, of a silvery sheen, or even pellucid; the coloring depends upon the amount of bile-pigment that enters into the stone's composition.

PATHOLOGY.—Gall-stones, in the vast majority of cases, are formed in the gall-bladder, occupying the cystic, hepatic, or common duct secondarily. Intrahepatic stones, a very exceptional variety, are formed in the intrahepatic ducts. A stone from its point of origin may escape from the body *per vias naturales.*

If we ascribe the pathogenesis of gall-stones to a bacterial inflammation, we should expect to find in all operations *in vivo* and in post mortem examinations the evidences of that inflammation; yet we know that gall-stones may be confined in the gall-bladder with no apparent pathological surroundings, and we further know that the gall-stones themselves may be sterile; as Mignot has shown, it is only from recent stones that cultures may be made. The initiating inflammation of the gall-bladder may have subsided, and the evidences of infection may have disappeared, yet in all cases in which there are marked symptoms we find cholecystitis or cholangeitis or both; and when such inflammations of the bile-passages exist, cultures are invariably successful in revealing microörganisms. In all cases of incarceration of the stone in the bile-ducts, dilatation of the channels above the point of impaction of the stone, with stagnation and alteration of their fluid contents, results; and furthermore, such obstruction and stagnation favors the entrance of microörganisms. In the etiology I have expressed my inclination to the hemic mode of infection rather than to ascension from the duodenum, the view that is almost universally held.

The effects of gall-stones, pathologic and symptomatic, immediate and remote, are influenced so largely by the location of the stone that they must be studied in its different habitations.

Stone in the Gall-Bladder: If the stone floats in its medium, the bile, untrammeled in the gall-bladder, the changes may be very slight, and it may escape from the body without causing structural alteration or symptomatic expression. It may be retained in the bladder because of its size, or the number, or from displacement of the natural relation that exists between the bladder and its duct. Their prolonged presence in the bladder leads to hypertrophy of the bladder-wall; this hypertrophy is both mechanical and inflammatory in nature, and results in contraction of the bladder around the calculus. The bladder-wall is thickened, is surrounded by adhesions, the result of pericholecystitis,

and in old cases it becomes calcareous from an incrustation of lime-salts upon the mucous membrane, and from an interstitial infiltration of the same deposit. Such an advanced change is, of course, rare, but all surgeons who have met with these thickened bladders will attest to their friability. The surrounding adhesions may contract and deform the bladder, or the stone within may erode the wall; cicatricial trabeculæ may form, imbedding the calculus and thus sacculating the bladder. Ulceration and perforation may occur. The rapidity with which these changes take place is not dependent upon the size or number of stones, but upon the virulence of the infection. If this be mild, the firm, thickened bladder may contract closely around the stone, and in this condition it may remain indefinitely; but at any time the element of active infection may precipitate acute changes.

Stone in the Cystic Duct: If the stone becomes engaged in the neck of the bladder or cystic duct the effects will depend in a great measure upon the completeness of the obstruction, upon the chronicity of the case, and preëxisting inflammation. There is no enlargement of the liver unless there is the unusual complication of cholangeitis, and jaundice is absent unless a stone of large size compresses the hepatic duct. In cases of long duration, with thickened bladder-wall and destroyed mucous membrane, there will be contraction of the bladder and practical obliteration of it; it will appear as a nodule, the size of a cherry or plum, or else as a cord-like cicatrix. The subsequent pathological changes in these cases are the result of pressure and inflammation produced by the stone. In more recent cases in which there is not material alteration in the bladder-wall, the effects of the imprisoned stone in the cystic duct will depend upon the virulence of the infection, and upon the completeness of the obstruction. Arménis has shown that it is possible for the bladder and its contents to remain aseptic in cases of obliteration of the cystic duct. It is conceivable under such conditions that the bladder might atrophy, but the process is usually otherwise in absolute obstruction. If the mucous membrane is not destroyed the bladder becomes distended with a colorless albuminous fluid, the secretion of epithelia and glands of the gall-bladder, and it is not bile from which the coloring matter has been absorbed; its accumulation leads to ectasia, the *hydrops vesicæ felleæ* first described by

Fantoni, producing a tumor which may vary from the size of a fist to that of a child's head or larger. This fluid may become infected, thus causing a simple empyema of the gall-bladder, or in virulent infection the most serious phlegmonous cholecystitis (Robson).

Much study has been given to the so-called ball-valve obstruction of the bile-ducts by calculi. Courvoisier, Fenger, and Lauenstein explain many of the physical phenomena by this action. In some instances of partial obstruction of the cystic duct, we find the bladder greatly distended with bile; in such cases, the obstruction permits the entrance of bile, but does not allow its discharge from the bladder. In other cases the opposite condition may exist: the obstructing calculus will prevent the entrance of bile, but will permit outflow of the bladder contents; in such cases the bladder may be a flaccid sac, but will ultimately undergo atrophy and contraction.

In either case of complete or partial obstruction of the cystic duct, should the stone retreat into the bladder, or escape by the channels into the intestine, the tumor will disappear by the discharge of its contents through the natural canal. There are two possible exceptions to this, however; the changed relation of the bladder to the duct, the result of overdistention and adhesions, may occlude the lumen of the cystic duct; or the stone may have excited an inflammation resulting in obliteration of the duct. The intermittent occlusion of the cystic duct may be mechanical or inflammatory; and the phenomena ascribed to ball-valve action of stones are probably due to inflammatory obstruction.

In obstruction of the cystic duct infection of the gall-bladder generally occurs, and an acute cholecystitis of varying intensity results, the contents of the bladder often becoming purulent.

Stones in the Intrahepatic Ducts: This condition is rarely met with. Courvoisier records several historical instances; in one case 30 concrements were found distributed throughout the liver-tissue; in another, two stones the size of a pigeon's egg were enclosed in a thick-walled cyst. It must be borne in mind that large stones found in the liver may occupy that position secondarily, their primary seat and origin being the gall-bladder. The intrahepatic concrements are usually gall-sand, or minute formations.

Stone in the Hepatic Duct: The occasional presence of stones in the hepatic duct is usually associated with calculi in the common and cystic ducts. They not infrequently are displaced by operative manipulation from the common to the hepatic duct. The effect of an obstructing stone in the hepatic duct is to produce dilatation of the ducts above the seat of obstruction, stagnation of bile, enlargement of the liver, and, with infection, cholangeitis, pronounced jaundice with rapid destruction of the red corpuscles, and an early fatal termination.

Stone in the Common Duct: According to Courvoisier, calculus is arrested in the common duct in about 4 per cent. of all cases of obstruction. Of 249 cases of cholelithiasis operated upon by Robson, in 13.25 per cent. gall-stones were found in the common duct. The latter figure, perhaps, more correctly indicates the frequency of this phase of cholelithiasis.

In the majority of cases the obstruction is produced by one stone. It is quite common to find numerous stones in the gall-bladder and a single large stone obstructing the common duct. In 97 cases of cholelithiasis, Conradi found stones both in the common duct and gall-bladder in 10 instances, and in the common duct alone in 5. Alessandri says that stones in the common duct are arrested in the duodenal portion in 67 per cent. of the cases. The stone may be freely movable within the duct, yet too large to escape from the papilla. The floating stone, as we are taught by Fenger and others, possesses a ball-valve action, to which mechanical feature and to the associate inflammation the varied pathological findings and symptoms are due. An arrested stone in the common duct does not necessarily produce an absolute obstruction to the outflow of bile, but it excites throughout the whole biliary channels vicious contraction to effect its expulsion; this leads to compensatory hypertrophy and dilatation, quite analogous to what is found in chronic intestinal obstruction. The relaxed distal portion of the duct, below the seat of obstruction, permits of the easy entrance of infection from the intestine, so that in all cases of chronic bile-duct obstruction by calculi, hypertrophy, dilatation, and inflammation are essential pathological elements. In those acute cases in which a stone suddenly enters the common duct and occludes its lumen, dilatation with thinning of the wall of the duct without hypertrophy takes place;

this ectasia, due to the damming back of the bile, may involve the entire system of biliary channels.

The dilatation of the bile-ducts, except in very acute cases, is not uniform; sacculation or a spindle-shaped dilatation at the seat of obstruction is more common. The ducts may exceptionally be so much enlarged as to be easily mistaken for an intestine. The hepatic duct has been found so capacious as to admit three fingers; the common duct may be so dilated as to form an abdominal tumor, and in several instances it has been mistaken for a distended gall-bladder, attached to the abdominal parietes and drained. Cholangeitis, simple catarrhal in character, is constantly present and may exist for years, or it may suddenly become suppurative, leading to diffuse suppurative hepatitis, abscess of the liver, general septicemia with metastatic abscesses, meningeal and endocardial lesions. The pressure of the calculus, aided by infection, results in erosion of the mucous membrane, ulceration and perforation. In all chronic cases there is inflammation of the structures around the duct, and adhesions are acquired leading to complications hereafter mentioned.

The most interesting part of common-duct obstruction by calculi is the attendant condition of the gall-bladder, which is usually in a state of atrophy and contraction. Fenger explains this by the ball-valve action of the stone, but Robson shows very clearly that it is found when the stone is impacted and immovable. There are several elements that account for the contraction of the gall-bladder: first, the chronic cholecystitis, with its consequent hypertrophy and thickening, excited by the stone while resident in the gall-bladder; second, during the passage of the stone through the cystic duct the gall-bladder is practically placed out of commission, atrophies and contracts; third, when the stone finally reaches the common duct, the obstruction rarely being absolute, the backward pressure of bile is insufficient to overcome the resistance offered by the rigid, contracted bladder; fourth, in many instances in which calculus is found in the common duct there are also stones in the cystic duct and gall-bladder, about which the organ has contracted; fifth, granting the hepatic origin of some calculi, the cholangeitis which invariably attends such formations will usually extend to the cystic duct and gall-bladder, resulting in changes similar to those produced

by a stone originating in the bladder, and to this inflammatory change the non-distensibility of the organ is due. It therefore follows that it is a significant clinical feature that common-duct obstruction by calculi is rarely attended by a gall-bladder tumor.

Enlargement of the liver would be conceded as a natural consequence of common-duct obstruction, and this would inevitably occur, if the obstruction was permanent and absolute; so, at first, there is no enlargement of the liver, but ultimately the organ may become enormously enlarged—a true biliary hypertrophic cirrhosis from bile-stasis. In acute cases with cholangeitis there is always enlargement of the liver.

The toxemic condition of the patient, as expressed by the emaciation and jaundice, is the most obvious, if not the most important part of gall-stone obstruction. Netter and Gilbert and Girode have found pyogenic organisms in cultures from the blood of patients with gall-stones; and where jaundice is marked, there is fragmentation of red blood-corpuscles, and there is marked decrease in their number, and decline in hemoglobin.

Complicating Pathology.—In the preceding pathology we have purposely restricted our attention to the changes in the biliary passages and liver, consequent upon the presence of gall-stones. At the risk of repetition, for the purpose of systematizing and simplifying, we now note the complicating pathology of gall-stones in their common sites.

In the Gall-Bladder: A stone in the gall-bladder, too large to engage in the cystic duct, invites infection by its irritation, leads to the development of cholecystitis and pericholecystitis; adhesions to the adjacent structures, especially to the colon and parietes, are acquired. Pressure-necrosis, aided by infection, will result in a perforation of the bladder-wall, the discharge of the stone into the intestine, or the formation of an abscess in the wall of the abdomen, through which the stone may be discharged; this is the most frequent mode of escape; Courvoisier has collected 184 cases, in one-fourth of which the opening was through the umbilicus. In other instances adhesions are insufficiently formed, perforation takes place into the peritoneal cavity, followed by all the pathology and symptoms of perforation-peritonitis. In exceptional instances the infection is so virulent that

gangrenous cholecystitis occurs. The stone may, by accident, erode the inner wall of the bladder, burrow into the liver, occasionally becoming encysted, but usually giving rise to hepatic abscess or, by perforation, to a subdiaphragmatic abscess.

In the Cystic Duct: The conspicuous pathological phenomenon in cystic-duct obstruction is the distention of the gall-bladder. Should infection occur, there is a marked tendency for the process to remain localized to the bladder itself, but a general cholangeitis or a phlegmonous cholecystitis, with the symptoms of local peritonitis and the evidences of constitutional infection, may follow. Such a distended gall-bladder may acquire adhesions to any of the abdominal viscera, and by ulceration and perforation discharge its contents through them. Fistulous communications are established with the colon, duodenum, small intestine, kidney, vagina, uterus, and urinary bladder. Graham, from a study of a series of cases, notes that the gall-bladder may rise up over the projecting margin of the liver and discharge its contents into the pleura or lung without perforating the liver-substance. Usually, however, the bladder acquires attachments to the anterior parietes, an abscess is formed, and external biliary fistula is established. If stones are free in the gall-bladder they make their way to the surface and are discharged with the pus; those calculi impacted in the cystic duct may remain indefinitely, but the evacuation of the contents of the gall-bladder relieves the *vis a tergo* and the stone recedes from its imprisoned position in the duct into the bladder, whence it is discharged—nature's suggestion to the surgeon for cholecystostomy.

As our pathology has shown us, cystic-duct obstruction is not always attended by ectasia of the gall-bladder. An impacted stone in the cystic duct with a contracted bladder has fewer chances to escape, and offers greater peril to the patient. By its size and attendant inflammation, it may obstruct the hepatic duct or common duct, giving rise to the complications that attend occlusion of these passages. It may fortunately find escape through an intestinal fistula, but more frequently acute infection with its cholangeal and hepatic inflammation develops. A stone that has passed through the cystic duct, in its passage may have done such injury to the duct that organic stricture remains. The adhesions surrounding an inflamed gall-bladder and its ducts.

aside from their immediate effects upon these passages, may lead to intestinal strangulation, or, at least, by anchorage render peristalsis difficult.

In the Common Duct: Dilatation and cholangeitis are the most marked local pathological changes resulting from gall-stone blocking the common duct. Cholemia is the apparent general effect. Enlargement of the spleen, pancreatitis, nephritis, endocarditis, hepatic cirrhosis, are common complications in other organs. Pericholedochitis leads to constriction of the duodenum and pylorus; Halsted records a case of dilatation of the first portion of the duodenum produced by constriction from adhesions for which a gall-stone was responsible. Influenced by infection, the local inflammation is more destructive; unprotected perforation may occur, resulting in general peritonitis or a circumscribed collection of pus. Pressure upon the vena portæ may lead to thrombosis, and cause capillary hemorrhage from the stomach or intestine. By erosion of blood-vessels fatal internal hemorrhage may occur. Hemorrhage from the biliary channels due to the altered condition of the blood in cholemia is reported as a fatal complication in gall-stone. The pancreas, either by pressure or ulceration, may suffer from the presence of a stone in the common duct. Smith reports a fatal case of impacted gall-stones with acute hemorrhagic pancreatitis. Acute hemorrhagic pancreatitis may be due to inflammation or to calculus-occlusion of the pancreatic duct with infection, or, Opie thinks and has proved experimentally, to the entrance of bile itself into the duct; Halsted's recent clinical observation led him to advance this theory which incited Opie's experiments.

A large stone escaping from the gall-bladder, cystic, or common duct, by fistulous connection with the intestine, very frequently produces volvulus or obturation of the intestine; it lodges usually in the ileum, and frequently in coils lying in the true pelvis. Lobstein collects 92 cases of intestinal obstruction from gall-stones; of 61 not operated upon 32 recovered and 29 died; of the 31 subjected to operation, 12 recovered. The entrance of the stone into the intestine is usually painless, through a slowly formed fistula. Ochsner records that of 18 patients operated upon for gall-stones 6 suffered from appendicitis; he thus expresses the significance of this complication: "I have

come to suspect the presence of chronic appendicitis in patients suffering from gall-stones." The bacterial origin of both affections is the only reason suggested for what I regard a mere coincidence. In the chapter upon Appendicitis, I have referred to a case in which I found a true gall-stone producing gangrenous appendicitis; this merely shows the accident that may result from the passage of a foreign body through the intestinal tract.

The association of gall-stone with primary carcinoma of the gall-bladder and biliary ducts is a subject of especial importance, and the reader is referred to the chapter on Primary Carcinoma of the Gall-Bladder and Bile-Ducts.

GENERAL SYMPTOMS.—The frequency with which gall-stones have been found at necropsy, not having produced symptoms during life, and the recognized clinical fact that practically every conceivable complication except malignant degeneration has been recovered from without surgical aid have, in the mind of the general practitioner, lessened the pathological dignity and minimized the dangers of gall-stone. The lost returns in those cases dying, labeled "internal congestion," "peritonitis," "cirrhosis of the liver," "heart-disease" and a host of other maladies unnecessary to mention, in which the essential pathogenesis was a biliary concrement, allows no estimation of its true frequency. Kehr, when asked why he met with so many operative cases of gall-stone, modestly ignoring his great renown in this field, replied that his confrères were skilled in its diagnosis and appreciated its dangers. The possibilities of surgery emanating from Bobbs, elucidated by Sims, and proved by Tait, awakened renewed interest in this long-recognized disease, and now the daily demonstration by surgeons of the cause and its effects has removed many of the erroneous ideas that befogged the symptomatology. The subject appeals alike to the student of internal medicine and of surgery, and no further stimulus is needed for its scrutinizing study.

Cholelithiasis is a *common disease*. It occurs in those of any age, in both sexes, and in all conditions of life. It is especially prone to appear symptomatically between the thirtieth and sixtieth years. Its greater prevalence in women who have borne children and in obese subjects are facts for consideration. The

symptomatic onset is usually so sudden that it is spoken of as "an attack of gall-stone," yet in not a few instances very definite premonitory symptoms occur. Constipation, flatulency, loss of appetite, vertigo, tinnitus aurium, migraine, urticaria, icteroid tinge and scanty, dark urine are symptoms indicative of an approaching attack, which, when it appears, is designated as biliary, hepatic, or gall-stone colic. The attending phenomena we shall separately describe.

Pain: The pain is intermittent, paroxysmal in character, and is usually of sudden onset, of moderate severity at first, intensifying with each recurring paroxysm until it reaches its climax, when it may suddenly subside. Much diagnostic significance is attached to the time of its occurrence, which is usually at night, during the relaxation of sleep; it is presumably excited by the passage of food through the duodenum. It may occur at any time; violent exercise, hard breathing, vomiting, coughing, sneezing, are some of the many precipitating causes. In the beginning the pain is usually located in the epigastric region, shifting thence to the right hypochondrium, and referred to the back under the angle of the scapula. Occasionally the abdominal pain may be slight, and a referred pain of greater severity in the thorax or right shoulder, very infrequently in the left, is felt. This pain is never referred down the arms, and rarely to the sacrum or hips. Change of posture, lying upon the face, or forced flexion of the trunk, and firm pressure over the gall-bladder may mitigate the suffering. The attack may last only an hour or two, or several days, with complete intermissions, or remissions with exacerbations, usually ending during violent emesis. In cases of great severity the pain is described as agonizing; patients lose all self-control, roll upon the floor in anguish, and women describe it as infinitely more severe than the pangs of labor. The intensity of the suffering is depicted in the countenance; there is marked depression, the breathing is short and hurried, a cold sweat appears upon the brow; shivering sensations or distinct rigors accompany this condition; the surface may be cold, though the central temperature may rapidly rise to 104°; the pulse is weak, usually slow, occasionally fast and feeble; epileptiform convulsions are not infrequent; in the agony of a paroxysm, patients have been known to die.

In seeking an explanation for this distressing phenomenon it stands as an undisputed fact that so long as the gall-stones are quiescent in the bladder they do not give rise to this symptom. The pain is generally regarded as a signal that nature is attempting to expel the concrement from the capacious bladder through the narrow biliary passages. It becomes engaged in the vesical end of the cystic duct, and immediately there is excited a contraction of the bladder; this muscular spasm is the most active element in the production of pain. Riedel, and lately Kehr attach the greatest importance to the inflammation as a cause of pain. The fact that gall-stones are innocent tenants of the gall-bladder so long as there is an absence of inflammation, and further, the clinical fact that pain is sometimes relieved by a course of alkaline waters, though no gall-stones may be passed, tend to confirm such a view. The presence of the stone in the duct as a foreign body, the muscular spasm, the associate inflammation, and the stagnated and accumulating fluid behind the stones, are the elements concerned in the production of pain. A small, smooth stone may make the transit of the entire biliary canal without exciting pain; larger stones of rough surface or angular shape cause greater suffering; yet it has been noted that in cases of biliary fistula, severe colic and referred pain have been excited by the mere introduction of a small, smooth probe.

Assuming that a stone is passing from the gall-bladder to the duodenum, its progress is said to be marked clinically by suffering of great severity as it traverses the cystic duct, then by milder paroxysms as it passes along the common duct until it reaches the papilla, where again the pain becomes severe, suddenly ceasing with the discharge of the stone into the duodenum. To assume thus that the relative size of the stone to the ducts governs the intensity of the pain is to make a false assumption; Courvoisier has shown that of the 9 cases of gall-stone reported in the literature, in which death occurred during the paroxysms of violent biliary colic, in 6 instances the stone was in the common duct, which is larger than the cystic duct, disproving the mechanical theory. Inflammation of the gall-bladder and of the ducts, with the surrounding peritonitis, exaggerates the sensibility, occludes the lumina, and hence adds much to the intensity of the pain; that stagnation and retention of the bile is an element in the

production of pain is shown in those cases in which the symptom of colic has led to the diagnosis of gall-stone, and operation has shown only viscid bile without occlusion of the lumina of the ducts.

Vomiting: Gastric disorders, nausea and vomiting, may occur as symptoms of gall-stone, without the colic; but with the onset of pain there is always marked nervous depression with a feeling of faintness and nausea; this usually results in vomiting in the height of the paroxysm. Temporary relief follows the relaxation that attends the vomiting, but with the return of the pain sickness again appears. It is a reflex nervous symptom when it appears during the paroxysms. The vomiting is first of the contents of the stomach, then of bilious matter; but though continuous and persistent at times, it never becomes stercoraceous. Not infrequently an attack of gall-stone suddenly ceases during emesis. In chronic cholelithiasis associated with inflammation and adhesions, vomiting occurs as a symptom of partial obstruction of the duodenum, with consequent dilatation of it and of the stomach. In such instances the vomiting occurs some hours after taking food, is copious and followed by immediate relief. Vomiting does not always subside with the successful passage or operative removal of the stone; Mayo Robson reports instances of death from persistent vomiting, days and weeks after a satisfactory operation. In complete obstruction of the common duct, the vomitus does not contain bile. The complications of cholecystitis and cholangeitis are marked by frequent nausea. With the occurrence of perforation and its consequences, vomiting without nausea is usually a persistent symptom. Blood may appear in the vomitus, the result of capillary oozing from the mucous surfaces, due to cholemia or to venous engorgement from thrombosis or cirrhosis. In the process of ulceration the large blood-vessels may be opened, giving rise to copious hemorrhage into the stomach or intestine; such cases usually have an immediate fatal termination.

Shock: The pain of biliary colic produces vital depression. In slight cases it is marked by simple weakness and a feeling of prostration; in others, shock of varying intensity appears; collapse and death have occurred during the height of the paroxysms. The nearness of the seat of trouble to the abdominal

nerve-centers, the rich nerve-supply to the biliary ducts and gall-bladder, the intensity of the pain, the associate vomiting, and mental emotion are contributing elements in the production of the phenomenon of shock.

Pulse and **Temperature**: The pulse is especially and peculiarly influenced. It is generally stated that in all cases of biliary colic the pulse is slow; there are too many exceptions for this to be regarded as a rule. A slow pulse does occur as the result of the disturbance of the sympathetic control, and as an expression of cholemia; the heart-beat may be reduced to 40, or even to 20 per minute. If there is infection, as expressed by rigor or chill, the pulse is usually accelerated and feeble. A persistently rapid pulse is of most serious import.

The temperature, owing to the vital depression in the paroxysm, may be subnormal. We are often misled by the cold surface, the rectal temperature generally showing elevation, and in cases of the arrest of stones, especially in the common duct, obstructing intermittently as a ball-valve, there is a great elevation of the temperature. In these cases the occurrence of chills, fever, and sweats is one of the most important and confusing symptoms of cholelithiasis.

Jaundice: This is a symptom of obstruction, retention and absorption of bile. It is not pathognomonic of cholelithiasis, and what is still more significant, it is not a constant symptom of this condition. From 1890 to 1902 laparotomy for gall-stone was performed by Kehr 720 times. Icterus was wanting in 80 per cent. of cases of stones in the gall-bladder and cystic duct, and was absent even in 30 per cent. of cases in the common duct. In the mind of the practitioner it seems deeply rooted that jaundice is associated with gall-stone, and that without jaundice the presence of gall-stone is doubtful, and its dangers insignificant. That such is not the case there is abundant evidence to show. Jaundice developing without pain means something other than gall-stone. The old formula of pain + jaundice = gall-stone no longer obtains.

In the prodromal stage of biliary colic slight icterus may appear. Pronounced jaundice is usually not evident during the paroxysms, but, as a rule, 24 to 48 hours after the onset of the pain. Slight icterus is not noticeable by artificial light; it ap-

pears first upon the right side of the abdomen, and in the areas of natural pigmentation, even before the scleral conjunctiva reveals the coloring. The face may retain its hue of health, and yet jaundice of the surface of the abdomen be quite apparent. The urine will reveal the presence of biliary pigments before the staining of the skin appears; the mere appearance of the urine is not sufficient evidence of the presence of bile-pigment. Gmelin's test should be employed for its detection. Marechal's iodin test is sufficiently accurate for clinical purposes. In deep jaundice the skin and mucous membrane eliminate the bile, and the perspiration will stain the clothing; the presence of bile in the saliva causes the bitter taste in the mouth. The serious cholemia may be evidenced by ecchymotic spots on the body and on the mucous membranes. In acute jaundice dependent upon common-duct obstruction, there is always the slow pulse above referred to. In chronic jaundice the anemia, emaciation, and debility are usually indicated by a rapid and feeble pulse. Grave nerve symptoms may occur if the icterus is prolonged; despondency, melancholia, stupor, delirium and coma, with dry, brown tongue, diarrhea and repeated vomiting, are the common manifestations. When the functions of the kidneys are much disturbed, muscular tremors and convulsions may ensue. The typhoid condition, the cardiac disturbance, the depression of the nervous system, are all symptoms of the auto-intoxication of cholemia.

The size and location of the calculus and the completeness of the obstruction determine the degree of jaundice; it, therefore, follows that a calculus obstructing the hepatic or common duct interrupts the flow of bile, the absorption of which then produces jaundice. A stone in the gall-bladder or cystic duct will not do so; exceptionally a very large calculus in the cystic duct may narrow by pressure the lumen of the hepatic duct and produce slight jaundice. Multiple calculi may permit the escape of bile more readily than a single stone acting as a ball-valve. Ordinarily in cholelithiasis the jaundice that follows the paroxysm slowly fades to recur in deeper hue with a return of the attack. Persistent jaundice usually means complete obstruction with cholangeitis, or else the graver condition of malignancy. The occurrence of slight jaundice after a severe attack of hepatic

colic does not necessarily imply impaction and obstruction by the stone in its transit through the biliary passages; it may be attended by sufficient inflammation of the mucous membrane of the biliary passages to cause temporary bile-stasis and icterus.

Skin: Simple pruritus or urticaria is a symptom common in hepatic trouble, more frequently in other diseases than cholelithiasis. It occurs without jaundice, hence the theory that it is due to the irritation of the bile eliminated by the skin does not fully explain this symptom; it is probably of nervous origin. With pronounced icterus, distressing itching and creeping sensations are felt over the surface of the body. A special eruption, known as *xanthelasma,* which is usually limited to the eyelids, but which in some instances extends over the whole cutaneous surface, composed of pale-yellow plaques or nodules, may occur in chronic cholelithiasis with jaundice.

Stools: Constipation with flatulency is one of the prodromal symptoms of biliary colic. Obstinate constipation with distention and vomiting is a constant symptom during the seizure, simulating intestinal obstruction. In cystic-duct obstruction the stools remain unchanged. Decolorization of the stools occurs only with complete obstruction; jaundice may be pronounced, yet stools showing the presence of bile evidence that the bile-stasis is not absolute. In intermittent obstruction due to ball-valve stone the color of the stools may vary from time to time. The evacuations become soft, mushy, pultaceous in character, and are described as pasty, putty-like; they resemble the familiar dish known as "apple-float." The absence of bile from the stools, persistent jaundice, without recurring attacks of pain, point to other obstruction than that due to gall-stone.

A diagnosis of gall-stone not confirmed by operation or autopsy is open to doubt, unless the calculus is found in the stool. After a paroxysm of biliary colic the stools should be washed through a sieve and careful search made for stones. The shape of the lithoid does not indicate its point of origin in the biliary passages, nor does the absence of facets prove that there are no other stones.

Urine: Unless there is jaundice the urine is not affected. Since the kidneys are the chief eliminators of the poison they soon suffer from irritation; the urine becomes deeply stained with bile-

pigment, resembles lye, albumin appears in it, urea is diminished, the total solids are much less than normal, and the quantity of urine voided at first is markedly increased, then diminished in amount, a signal of great danger. Scanty urine in marked jaundice is a precursor of convulsions and death. Naunyn, in 250 cases of gall-stone colic, never found glycosuria except in those cases in which there was complicating disease of the liver. Even where there is ulceration with pressure upon the hepatic duct, sugar is not present.

PHYSICAL SIGNS.—**Tenderness**: The inflammation, causal or secondary, which attends gall-stone, usually by contiguity involves the surrounding structures; to this we attribute the physical sign of tenderness. Gall-stones resident in the bladder without cystic-duct obstruction give rise to tenderness over the viscus. With the development of acute cholecystitis this definite point-tenderness at the tip of the tenth rib assumes the greatest significance. In obstruction of cystic duct with chronic and contracting inflammation and pericholecystitis, tenderness is a constant and valuable diagnostic sign. In cholangeitis due to common-duct obstruction the tenderness is more diffuse and may cover the hepatic area.

Tumor: Gall-stone gives rise to enlargement of the gall-bladder and to an appreciable tumor under the following conditions: (1) In chronic cholecystitis with surrounding inflammation and adhesions of intestines and omentum; such a tumor is flat, irregular, ill-defined, palpable but not visible. (2) In acute obstructions of the cystic duct preceding which there has not been inflammation and contraction of the gall-bladder, the walls of that viscus retaining their normal elasticity; this tumor is a typical gall-bladder enlargement. (3) In acute obstruction of the common duct, not accompanied by occlusion of the cystic duct nor preceded by inflammation of the gall-bladder—an infrequent condition.

It appears, therefore, that a tumor is a sign of acute calculus-obstruction rather than of chronic, that it follows occlusion of the cystic duct, and is rarely developed as a consequence of common-duct obstruction. In obstruction of the cystic duct distention of the gall-bladder occurs either as a hydrops or as an empyema; in ball-valve obstruction the contents may show an

admixture of bile. As the viscus enlarges it advances downward and forward from out the anterior portion of the external longitudinal fissure of the liver, its normal position. When moderately distended the swelling lies between the right parasternal line and the anterior axillary line, a little to the right of the right rectus muscle. We must not forget the possibility of transposition of viscera; Beck records a case in which the liver occupied the left side, gall-stones being removed from the bladder by an incision at the outer border of the left rectus muscle. The tumor is ovoid or pear-shaped, its long axis pointing inward towards the foramen of Winslow. In great distention of the gall-bladder the tumor may occupy a large portion of the abdominal cavity; it rarely attains to a size larger than a child's head, and, as usually met with, is the size of a large pear. In obese subjects great distention may be present without a visible tumor.

Pollatschek suggests a method of palpation for the detection of an enlarged gall-bladder: place four fingers of the left hand over the edge of the liver lightly, and give necessary pressure with four fingers of the right hand placed upon the dorsal surfaces of the left fingers. The tactile sense of the palpating hand is thus preserved, and the smooth, globular, elastic fundus of the gall-bladder is felt in immediate apposition with the abdominal wall. Irregularities may be due to adhesions, or to sacculation of the bladder from stones. When the bladder is filled with stones and its walls are thickened, the tumor is densely hard and suggests malignancy. Fluctuation is not detected in distention of moderate degree.

The tumor responds to the movements of respiration. It may be displaced laterally, in some instances even to the left of the umbilicus. It may be pushed backward, but will not disappear into the loin. Its connection with the liver may be established, and if sufficiently pendulous, its lower end will move in the arc of a circle about the fixed point at the liver. Calculi can rarely be felt; in 200 cases Courvoisier was able to elicit the gall-stone crepitus only once. Efforts should not be made to elicit this; accidents of rupture of the bladder and of displacement of the stone to a more dangerous position have followed rude manipulation. Change of posture of the patient to the side or to the

knee-and-chest position may render palpable a distended gall-bladder not detected in the ordinary dorsal position. When the abdominal wall is thin we may be able to make out the linguiform extension of the liver—Riedel's lobe—by its sharp edge lying transversely across the upper part of the tumor, its smooth surface continuous with the liver. This sharp projecting edge of the liver, partly covering an elastic tumor, is pathognomonic of gall-bladder enlargement.

The tumor is dull upon percussion, and its continuity with the liver is established by percussion. Exceptionally it may be resonant, underlying adherent coils of intestine. Inflation of the stomach displaces the gall-bladder to the right. Aspiration needs only to be mentioned to be condemned.

It must be borne in mind that, following an attack of colic and sometimes without any known reason, a distended gall-bladder may spontaneously discharge its contents and the tumor disappear. Calculus-obstruction, giving rise to distention of the gall-bladder, is preceded by or accompanied with paroxysmal pain. A painless enlargement rather indicates malignancy.

Gall-stone, though infrequent in children, occurs sufficiently often for the possibility to be taken into consideration. To elicit tenderness, or to detect a tumor in infancy, Wendel makes the admirable suggestion of having the nurse place the child in a warm bath, soap its head well; the infant will keep its eyes closed voluntarily, and with its attention diverted the surgeon may examine the relaxed abdomen.

Enlargement of the Liver: Calculi in the gall-bladder or cystic duct do not give rise to bile-stasis, consequently there is no enlargement of the liver in such cases unless there is cholangeitis. This is somewhat contradictory to the observations of Fürbringer, who, in a study of 64 cases of cholelithiasis, says that there is almost always enlargement of the liver. In hepatic- and common-duct obstruction of the acute form the enlargement of the liver is due to the damming back of bile, and to the associate inflammation. Following chronic obstruction we have biliary hypertrophic cirrhosis. Calculi imbedded in the hepatic structure may produce irregularities upon the surface of the enlarged liver, and if suppuration occurs, the upward enlargement of a solitary abscess.

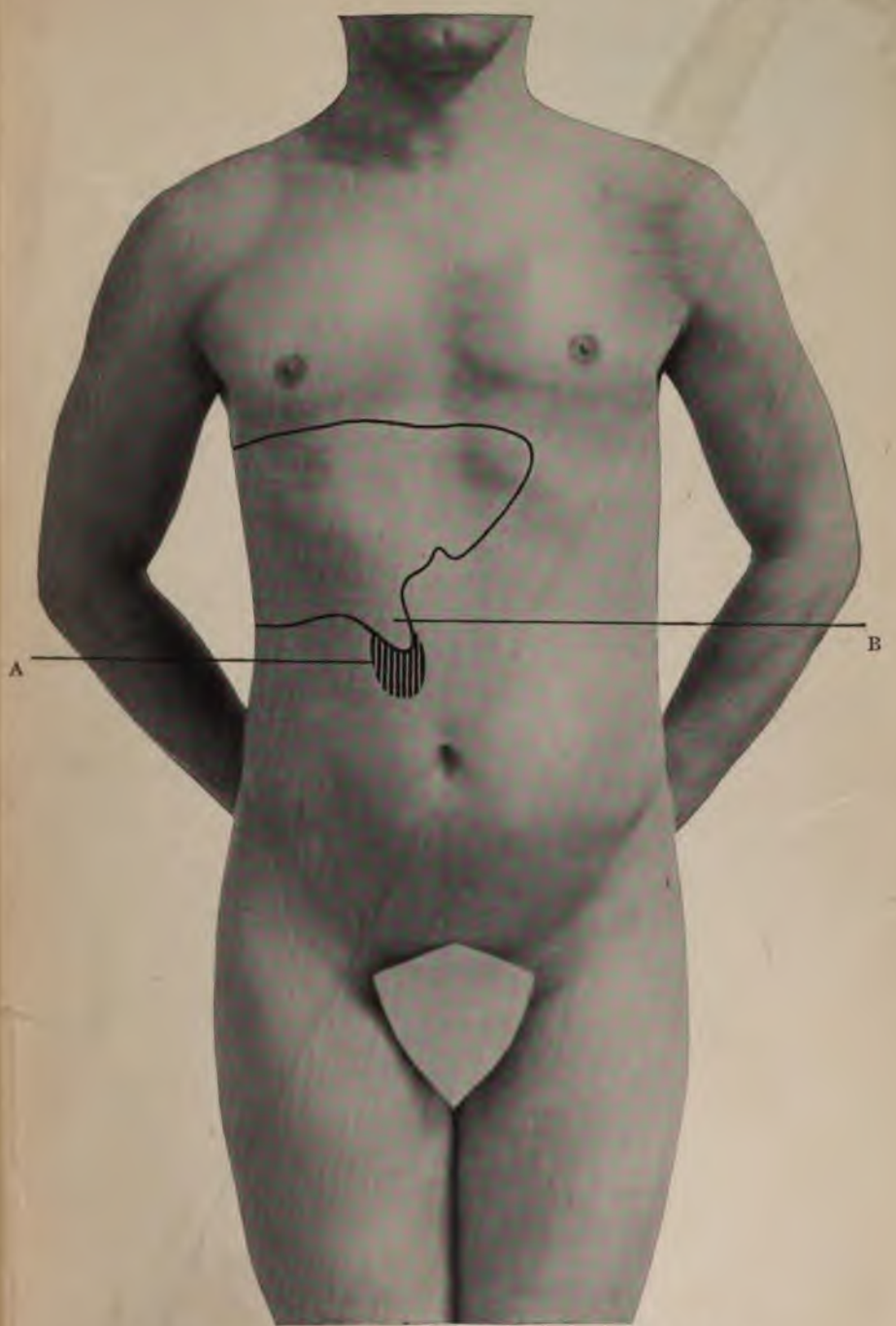
The Röntgen ray has proved of but little value in the detection of gall-stones.

In the Gall-Bladder: Calculi may remain in the gall-bladder an indefinite time without producing symptoms. Usually, however, their presence is associated with erosion of the mucous membrane, which opens an avenue for infection. The symptoms are then more attributable to the cholecystitis and its complications than to the mere presence of the stone. It is asserted that colic is only awakened when the stone becomes engaged in the cystic duct. That hepatic colic may result simply from spasmodic contraction of a gall-bladder due to inflammation, without the presence of a stone, is proved by ample pathological evidence, yet it is true that pain, but not colic, is a symptom of calculus of the gall-bladder. Local tenderness over the gall-bladder is rarely absent, and if there is associate cholecystitis, this point-tenderness at the tip of the tenth rib is equal in diagnostic value to McBurney's point in appendicitis. If we place one hand on the left side and the other over the right side and forcibly compress the liver, pain will be felt in the region of the gall-bladder. A sharp thrust with the tips of the fingers, Lloyd's "liver-bump," will cause a stabbing, lancinating pain. There is no enlargement of the gall-bladder unless there is obstruction of the cystic duct. A tumor, however, may be felt, attributable to adhesive pericholecystitis; this is rather an area of induration than a distinct tumor. There is no jaundice or evidences of bile-stasis, though the liver may be enlarged. The indefinite symptoms of digestive disorders, constipation, and nervous phenomena are usually present. Fever occurs with infection.

LOCALIZATION OF GALL-STONES.

In the Cystic Duct: The symptoms and physical signs, the outcome of impaction of stone in the cystic duct, depend upon the chronicity of the cholelithiasis, upon the acuteness of inflammation, and upon the completeness of the obstruction. With engagement of the stone in the cystic duct there occur the typical symptoms of hepatic colic. The obstruction offered by the small lumen of the cystic duct and Heisterian valves causes the smallest stone to excite muscular spasm in its efforts to pass. The paroxysms recur until the stone successfully passes to the intestine, or until it recedes into the gall-bladder. Colic, then, is the

PLATE V.



POSITION OF TUMOR AND ITS RELATION TO LIVER HYDROPS FELLEÆ.

A. Distended gall-bladder.
B. Reidel's lobe of the liver.

primary and important symptom of calculus-obstruction of the cystic duct. The attack is usually accompanied by vomiting, vital depression, and constipation. A chill and fever are not unusual. There is no jaundice, yet in these cases careful urinary analysis may show biliary pigment. If there has been calculus-disease of the gall-bladder with inflammation, existing for some time, then the viscus becomes thickened, contracted, and unyielding, a condition which will give a very different objective symptomatology from an acute obstruction with a dilatable gall-bladder. It must, therefore, be borne in mind that under some conditions of cystic-duct obstruction there may be an absence of a tumor; but it cannot be said that it is the habit, although it is the occasional behavior of gall-stones to remain long quiescent in the gall-bladder, and hence usually in the obstruction of the cystic duct there is a dilatable gall-bladder and consequently a tumor. If the occlusion of the duct is absolute no bile can enter, and the gall-bladder becomes filled with its own secretion, *hydrops vesicæ felleæ*. If there is infection from pyogenic germs the fluid becomes purulent, and we have empyema of the gall-bladder. If the obstruction is incomplete, bile may be admitted to the bladder and be prevented from escaping; the fluid distending the gall-bladder would then probably be a mixture of pus, bile, and mucus, and the tumor would rapidly form. The usual phenomena of cystic-duct obstruction are paroxysms of pain, vomiting, possibly a chill and fever, though this is frequently absent, local tenderness, and all symptoms may suddenly subside; or, if the obstruction remains, a tumor gradually forms. There is no jaundice. If the stone becomes impacted permanently the paroxysms of pain subside, the vomiting ceases, but tenderness remains and a tumor may develop.

In the Common Duct: Important in the symptomatology of calculus-obstruction of the ductus choledochus is the history of previous attacks of biliary colic. It is true, the primary evidence of the presence of stone may appear as an obstruction to the common duct, but in its route from the liver or from its almost invariable seat of origin, the gall-bladder, it does not reach the common duct without giving rise to colic. There are three elements concerned in the symptomatology of obstruction of the common duct: pain, bile-stasis, and inflammation. The pain is more in-

tense, if possible, than in cystic-duct obstruction, and oftener referred, especially to the back, more prolonged, and productive of greater shock. Vomiting is more obstinate, fever is usually present, and rigors are common. Jaundice develops within 24 to 36 hours after the onset of pain; the urine becomes quickly and deeply colored.

Further symptoms depend upon special conditions. If the stone becomes firmly impacted in the common duct the icterus deepens, the stools are decolorized, the liver enlarges, and the symptoms of cholemia, the lethargy and extreme prostration, are marked; for reasons repeatedly given there is no enlargement of the gall-bladder. If the stone is not impacted, but partially obstructs the common duct and is movable in the ampulla of Vater, the condition becomes chronic, and the occasional efforts of nature to expel it through the papilla produce recurring attacks of colic, jaundice, chills, fever, and sweats, with or without clay-colored stools. This is the *fièvre intermittente hepatique* of Charcot, the symptomatology of which is thus briefly and accurately given by Osler: "(1) Jaundice of varying intensity deepening after each paroxysm, which may persist for months or even years. (2) Ague-like paroxysms, characterized by chill, fever, and sweating, after which the jaundice usually becomes more intense. (3) At the same time of the paroxysm, pains in the region of the liver with gastric disturbance." The interesting feature of this condition is the presence of the symptoms of chills, fever, and sweats, without suppurative inflammation of the bile-channels or other lesions than dilatation and mild catarrhal cholangeitis. The greatest confidence by all surgeons engaged in this field of work is accorded to this group of symptoms as diagnostic of a movable stone in the common duct and cholangeitis.

It is asserted by many, by Brewer especially, that all symptoms disappear in the intervals between paroxysms. I have seen four typical cases of this form of common-duct obstruction, three proved by operation; these patients were under observation many months, and the intervals between paroxysms would vary from two weeks to two months; bile was always present in the urine, the icteriod hue never entirely disappeared from the abdomen, and there was constant hypogastric discomfort or backache.

Common-duct obstruction is frequently associated with stones in the gall-bladder, and perhaps in the cystic duct; there are no means by which we can diagnosticate calculi in dual positions.

In the Hepatic Duct: Calculi occupy the hepatic duct secondarily. They are usually associated with calculus-obstruction of the common duct, and frequently with multiple calculi of the gall-bladder. The duct has been found packed with stones up to its bifurcation. Of fifty-nine cases of calculi in the hepatic duct collected by Courvoisier, fifty-six had stones in the gall-bladder and common duct as well. The symptoms are not specially diagnostic, and differentiation cannot be made from common-duct obstruction. There is atrophy of the gall-bladder, enlargement of the liver, jaundice, paroxysmal pain, remitting but not entirely intermitting, and usually fever from cholangeitis, which may become suppurative in character, leading to multiple hepatic abscesses and pyemia.

In the Intra-hepatic Ducts: Stones in these ducts cannot be diagnosticated *in vivo*.

DIFFERENTIAL DIAGNOSIS.—To assert that the general diagnosis of gall-stone is easy, and that the position of the calculus may in all instances be definitely determined is an acknowledgment of unfamiliarity with the manifold intra-abdominal pathological lesions which often present common and confusing symptoms. If the surgeon wishes to avoid the mortification of an erroneous diagnosis, and to save his patient from the consequences of an improper and, perhaps, needless operation, he must not rest content with the diagnosis of biliary calculus until he has excluded those conditions which closely simulate it. An indefinite conception of the true pathological state in all surgical work begets imperfect and prolonged technic, which influences materially the result. The differential diagnosis, therefore, becomes a most important part of the study of cholelithiasis.

Gastralgia: The paroxysms of gall-stone are frequently mistaken for gastralgia—I use the term in its restricted sense as expressive of a gastric neurosis.

Gall-stone Colic.	*Gastralgia.*
Type of Patient.—Common in obese, phlegmatic individuals.	In neurotics and hysterical patients.

Gall-stone Colic.	*Gastralgia.*
Time of Pain.—Usually at night; probably not affected by ingestion of food, certainly not relieved by it.	When stomach is empty, may show daily periodicity; relieved by food and by warm drinks.
Seat of Pain.—At first in epigastrium, region of gall-bladder, referred to back and shoulder.	Epigastrium or left hypochondrium, referred to left thorax.
Vomiting.—Copious and recurring.	Nausea, but vomiting slight.
Jaundice.—May be present or develop in a short while.	None.
Chill.—Frequently attends paroxysm.	None.
Fever.—Usually some elevation of temperature, pyrexia may be marked.	None.
Pulse.—Often abnormally slow.	Frequent, feeble, may be irregular.
Tenderness.—Definite local tenderness.	None, pressure affords relief.
Tumor.—May be present or develop.	None.

Gall-stone Colic.	*Gastric Ulcer.*
Age.—After 30 years.	Before 30 years.
General Health.—May be good.	Anemic, with tuberculous tendency.
Pain.—Paroxysmal.	Intermittent or constant.
Seat of Pain.—Right hypochondrium, and under angle of right scapula.	Left epigastric triangle or left hypochondrium; referred to back.
Time of Pain.—Usually at night.	Immediately after taking food.
Vomiting.—During paroxysms of pain.	Until stomach is relieved of its contents.
Hematemesis.—Rare.	Common.
Fever.—Frequently present.	None.
Chill.—Occurs in paroxysms.	None.
Jaundice.—May be present.	None.
Stools.—May be decolorized.	May show melena.

Gall-stone Colic.	*Gastric Ulcer.*
Tenderness.—Over gall-bladder.	Two inches to left of 12th dorsal vertebra, and over seat of ulcer.
Muscular Rigidity.—May exist, of right rectus.	Usually present, of left rectus.
Tumor.—May be found in region of gall-bladder.	None.

Gall-stone Colic.	*Intestinal Colic.*
History.—Previous attacks, perhaps with fever, chill, and jaundice; calculi may have been detected in the stools.	Recent errors in diet or exposure to cold.
Onset.—Sudden.	Prodromal symptoms of gastric distress, flatulency, etc.
Seat of Pain.—Epigastrium or right hypochondrium.	Umbilical region.
Vomiting.—May not occur until climax of pain is reached.	May precede the pain and accompany paroxysms.
Bowels.—Constipation.	Diarrhea or tenesmus during seizure.
Tenderness.—Over gall-bladder.	None, pressure is voluntarily made.
Muscular Cramps.—None.	May be general.
Tumor.—May be present.	None.
Duration of Attack.—May last an hour or a week.	A few hours.
Mode of Relief.—Spontaneously by passage or dislodgment of stone.	Free bowel evacuation of stool and flatus.

Gall-stone Colic.	*Acute Pancreatitis.*
History.—Previous attacks common.	None such.
Sex.—In women most frequently.	In men chiefly.
Onset.—Sometimes gradual, reaching a climax.	Sudden and violent.
Pain.—Distinctly paroxysmal.	May have exacerbations but no remissions.

Gall-stone Colic.	*Acute Pancreatitis.*
Seat of Pain.—Epigastrium and hypochondrium, referred to back under angle of right scapula.	Epigastrium, left hypochondrium and thorax.
Jaundice.—Common.	Infrequent. Cyanosis.
Vomiting.—May not occur except in paroxysms.	Persistent.
Shock.—Sometimes marked, but reaction is prompt.	When it appears usually persists to the end.
Urine.—Shows bile-pigment.	Negative, occasionally glycosuria.
Tenderness.—Over region of gall-bladder.	Deep-seated, epigastrium and left hypochondrium.
Tumor.—In gall-bladder region.	Not frequent; if found, in epigastrium or left hypochondrium.
Ascites.—None, ordinarily.	May develop early.

Movable Kidney: Perhaps the most confusing of all conditions connected with gall-stone is the differentiation of a distended gall-bladder from movable right kidney. They have so many features in common that a most painstaking differentiation is required. Both affections are more common in women who have borne children; in both, the patients suffer from dyspeptic symptoms, flatulency and nervous depression; both give a history of previous attacks of pain in the right side; in both there may or may not be slight jaundice; vomiting in the height of suffering is common to both conditions; a chill and fever may occur in both; a tumor, movable and situated in the right hypochondriac, lumbar, or umbilical regions, occurs in both; the tumor may be elastic, freely movable, and easily palpable; irregularities on the surface of the tumor occur in both conditions; the tumor in either case may be dull or resonant; the shape of the tumor made by a distended gall-bladder may closely resemble that of a kidney. Yet there are essential points of difference.

Distended Gall-bladder.	*Movable Kidney.*
Subjects. — More common in fat women over forty years.	More common in thin, neurotic women under forty years.

Distended Gall-bladder.	*Movable Kidney.*
Seat of Pain.—Pain of biliary calculus is in the epigastric or hypochondriac region, referred to the back and shoulder, always upward, rarely downward to sacrum or thighs.	In the lumbar, right hypochondriac, umbilical or inguinal regions, thence downward to sacrum, genitals and thighs.
Urine.—Scanty and high-colored.	May have suppression, polyuria, or ischuria.
Seat of Tumor.—In close apposition with anterior parietes; visible in thin subjects when of moderate size. By bimanual palpation tumor not readily felt with posterior hand.	Deeply situated, not visible. Easily felt between the hands.
Movement with Respiration.—Moves with respiration, will ascend with expiration.	May not be influenced by respiration; if grasped, will not ascend with expiration.
Anchorage of Tumor.—To the liver at the right parasternal line, lower and moving in arc of a circle about this as center.	No anchorage and no limitations in its movements.
Mobility of Tumor.—May be moved upward and laterally, rarely backward, never downward.	May be moved in every direction, and caused to disappear into the loin.
Shape of Tumor.—Usually pyriform or oval—may be sausage-shaped.	Kidney-shaped.
Resistance of Tumor.—A thickened gall-bladder filled with stones gives a hard, resistant tumor.	More yielding and elastic.
Percussion.—Dull, and dulness is continuous with that of liver.	Resonant over some part of its surface, and has a band of resonance between it and the liver-dulness.
Inflation of Colon.—Pushes gall-bladder upward, but will not efface its dulness.	May efface entirely the dulness.
Inflation of Stomach.—Moves gall-bladder to the right.	Causes to disappear entirely.

Distended Gall-bladder.	*Movable Kidney.*
Pressure of Tumor.—Elicits tenderness.	Causes faintness or nausea.
Pyrexia.—Common.	Unusual.
Riedel's Lobe.—Sharp edge frequently felt across gall-bladder.	Not so.
Auscultation.—Fremitus may occasionally be felt and heard.	A bruit sometimes audible over inner and upper border of tumor.
Disappearance of Tumor.—May disappear without extra flow of bile.	If there is hydronephrosis, disappearance marked by copious discharge of urine.

Carcinoma of the Gall-bladder and Bile-ducts: The subjects of cancer are usually over forty years of age. They show early cachexia. Pain may be absent, but is usually present, and is more constant and bearable than that of gall-stone. Jaundice may not develop at first, but when it appears it continuously deepens and does not clear up. The tumor of cancer of the gall-bladder is hard, irregular and nodular, fixed by adhesions, and is usually very tender. Enlargement of the liver occurs with the development of jaundice. Edema of the lower extremities and ascites are common phenomena. In old persons gradually deepening jaundice, with or without pain, with or without a tumor, should arouse a suspicion of malignancy.

Appendicitis: Nausea and vomiting usually precede the pain or accompany its mild onset. The pain is felt in the epigastric or umbilical regions at first, and subsequently is referred to the appendiceal region; it rarely intermits entirely, and does not subside with copious vomiting; it is increased by flexion and extension of the thigh. Tenderness develops early at McBurney's point. Muscular rigidity is present in more than half the cases. A tumor develops in eighteen to thirty-six hours frequently. In perforative or gangrenous appendicitis in which there is no tumor, much reliance may be placed upon the age of the patient, the history of previous attacks, the seat of suffering at that time, the rapid development of tympany, and the effacement of hepatic dulness. There is no jaundice, there is no pain on costal com-

pression; there is no definite point-tenderness at the tip of the tenth rib in appendicitis.

Malaria: A movable calculus in the common duct, creating the symptoms known as intermittent hepatic fever, is only too frequently mistaken for malaria. The negative results obtained from quinine, and the blood-examination showing the absence of Laveran's hematozoa, and the irregularity of the recurrences should eliminate malaria.

PROGNOSIS.—Neither the passage of gall-stones *per vias naturales,* nor their long residence in the gall-bladder and innocent behavior in instances can qualify the statement that cholelithiasis is a grave disease. When a calculus awakens symptoms sufficient for diagnosis, the condition must be regarded as one of imminent danger. One need only appreciate the possible occurrence of the complications, so repeatedly emphasized in this article, to realize fully the necessity of active interference. The detection of stones in the stools with subsidence of symptoms warrants only watchful delay. There are no statistics giving the percentage of mortality due to gall-stone—there can be none except in the limited number of cases that reach the hospital. The prognosis in any given case depends, first, upon the site of the stone; second, upon the attendant complications. In a general way we may say that a movable stone in any location is less serious than an impacted one; that the complication of jaundice by its pernicious influence on the blood and excretory organs of the body introduces new elements of danger. The occurrence of cholangeitis with enlargement of the liver and pyemic symptoms is the most serious of all conditions. Cystic-duct obstruction with distention of the gall-bladder is the most favorable case for operative relief. A guarded prognosis must be given in those cases of intermittent hepatic fever due to movable stone in the ampulla. The deepest jaundice and violent general symptoms may almost entirely disappear; upon the other hand, in any attack impaction with complete obstruction may occur, which, if not relieved, will lead to an early fatal termination.

TREATMENT.—**Medical:** The most that can be expected from the medical treatment of gall-stone is the establishment of a latent stage, and this can only be achieved when the stones are confined to the

gall bladder. The possibility of exerting any solvent action upon stones already formed is remote indeed. In fact, there is no rational grounds for believing in such a possibility. Just as improbable is the chance of encouraging the escape of stones from the gall bladder by medication. Massage might accomplish this, but under any circumstances it would be malpractise to do so. The likelihood of a stone, once forced from the gall bladder, passing through the cystic or the common duct is not great enough to justify such a procedure.

Alkaline waters, sodium salts (oleates, glycocholates, succinates, etc.), oilve oil and other vaunted solvents are but placebos which satisfy the patient and lull the conscience of the medical attendant. Rigid hygiene with any or no medication will often establish, as stated above, a latent stage when the stones are confined to the gall bladder. The duration of this period of latency is of course indeterminate, and the patient carries with him a condition which in spite of the utmost care on his part may at any moment become complicated and dangerous. If he thoroughly understands this risk, and prefers it to the risk of an interval operation, it is his privilege to make the choice. The case should be put fairly before him, however, and he should not be misled with any hope of actual medical cure.

Surgical: Billings states that gall-stones, when a diagnosis is surely made, is a surgical condition. The mechanics of the situation and the results of surgical treatment justify this position. Interval operation for stone in the gall bladder is little more difficult and probably no more dangerous than an interval appendectomy. The same comparison holds good in the presence of infection. With stones in the cystic, and particularly with stones in the common and hepatic ducts, the risk is greater, but surgery is unavoidable. By surgery alone can the patient be relieved, and there is not even hope of establishing a latent stage through hygiene or medication. In common and hepatic duct stones, with pronounced jaundice following a typical cholangeic paroxysm, it might be well to wait a few days for the jaundice to clear up, particularly when the history of the case justifies the assumption that it will do so.

When we come to actual operative procedures, our choice in the treatment of stones in the gall bladder itself lies between cholecystostomy and cholecystectomy. There is great difference of opinion among surgeons as to the indications for the removal of

the gall bladder, many preferring it as a routine procedure, while others only perform it when the gall bladder is hopelessly damaged, and not only useless, but dangerous if left in place. After cholecystectomy it is undoubtedly true that need might arise for the gall bladder for the purpose of establishing a collateral circulation, so to speak, for the bile. It is also true that cholecystectomy prevents many post-operative complications in the way of adhesions, fistulæ, further stone formation, possible malignant changes, etc. Each surgeon's opinion is to some extent based upon sad experience on one or the other horn of this dilemma. Aside from malignant disease, however, there will seldom arise the necessity for cholecysenterostomy, and we should not shape our surgery out of consideration for a condition that is in itself hopeless when it occurs. Cholecystectomy offers the best prospect for permanent cure, whenever there is well-marked inflammatory change in the wall of the gall bladder, whenever there is a damaged cystic duct, whenever pericholecystitis is present, and whenever the necessity arises for common and hepatic duct drainage. This last cannot be accomplished without resulting adhesions to the gall bladder, and these often give rise to great annoyance. Robson fears back pressure upon the liver and compensatory dilatation of the bile channels after cholecystectomy, and yet his indications for the operation leave little room for objection on the part of the most ardent cholecystectomist.

Cholecystectomy when done should be followed by drainage of the hepatic duct through an opening made by splitting the cystic duct down to the common duct, and then incising the common duct to such an extent as is required.

Common and hepatic duct stones require choledochotomy. Direct drainage, preferably of the hepatic duct, if the incision is suitably located, should always be used after this operation. When a common duct stone is in or near the ampulla, the duodenum should be opened and the papilla dilated or incised. This method of approach is better than the retroduodenal route.

The most essential features of gall-stone surgery are to insure removal of all common and hepatic duct stones and to provide ample drainage.

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CHOLECYSTITIS.

DEFINITION.—An infective inflammation of the gall-bladder.

FREQUENCY.—As a complication of other conditions, inflammation of the gall-bladder has long been recognized, but as a pathological entity, dissociated from the ordinary causes, it has only recently received consideration; and yet Maurice Richardson declares that it is more frequent in his experience than many intra-abdominal lesions, even more so than intussusception or volvulus.

ETIOLOGY.—Cholecystitis is a common associate of gall-stones; in its catarrhal form we have accorded it due prominence as a potent factor in their formation. We are chiefly indebted to Osler, Richardson, and Cushing in this country for a scientific explanation of the well-recognized clinical fact that cholecystitis occurs as a sequel of typhoid fever. The bacilli are constantly found in the gall-bladder during and oftentimes for years after typhoid infection. Hunner records a case of their isolation and culture eighteen years after an attack of typhoid fever. It is an open question how the infection takes place, whether by ascension through the common duct, or hematogenously, as Cushing maintains. The bacilli retain their vitality and show an intravesical agglutination; the clumps thus formed presumably become the nuclei of gall-stones. Both the diplococcus of pneumonia and the typhoid-bacillus are met with in the gall-bladder without the accustomed lesions of those diseases. An infection of this character may remain latent for years, and whether calculi are produced or not, the viscus involved ever after remains vulnerable and especially inviting to renewed infection. If the infection is mild it may be assumed that cholelithiasis is the common sequence. In more acute cases a phlegmonous or gangrenous cholecystitis may develop without the complication of calculus. That there are cases of acute cholecystitis, designated by Robson as 'phlegmonous,' and by Richardson as 'acute accidental cholecystitis,' which spring into existence entirely independently of any previous complaint or known cause, we are just beginning

to realize. The pathogenic agent seems to be the colon-bacillus, although it is suggested that other organisms may have existed, or even have initiated the trouble, and have been displaced by the colon-bacillus.

PATHOLOGY.—Suppurative cholecystitis—empyema of the gall-bladder—and simple catarrhal cholecystitis are expressions usually of mild infection pursuing either a subacute or a very chronic course, and are essentially different in their pathological manifestations from the more malignant inflammations of a phlegmonous or gangrenous character. The demonstration of microörganisms in the milder grades of inflammation is not always an easy matter, yet we cannot believe that they are absent, though they may not be found. Furthermore, the symptoms are due in a great measure to calculi which are generally present in these less virulent conditions; yet any chronic obstruction of the cystic duct may give rise to an empyema. In the malignant inflammations the symptoms are those of a septic infection. Having sufficiently considered under the subject of Gall-Stone its associate pathology, we are now more concerned with those cases, acute in character and destructive in tendency, not necessarily associated with calculi.

These inflammations, sudden in the onset, have pathological characteristics: a distention of the gall-bladder, as Courvoisier calls it, 'acute progressive empyema,' with an inflammation of the wall of the bladder, surrounding peritonitis, ulceration, perforation or gangrene of the viscus. Richardson thinks that the mere distention is an important pathological factor, which, carried to an extreme degree, may cause total gangrene. The course of this pathological process is exceedingly rapid. In a few hours from the initiation of the more fulminant cases perforation or gangrene may occur. In less virulent infections the progress may not be so rapid.

SYMPTOMS.—The onset is usually sudden and is characterized by sharp paroxysmal pains in the region of the gall-bladder or epigastrium. The pain is remittent in character; at first it may be diffuse and then localized, associated with great tenderness over the gall-bladder, so that Richardson says that the sensation and progress of these cases is that of an abscess under the right costal margin. The violent onset is attended by nausea, vomiting, and

great prostration. The pulse is usually rapid; the temperature, though generally high, may be misleading. A chill may occur with the initial symptoms or some hours later. There is no jaundice. Muscular rigidity and tenderness are especially characteristic features. There may be no tumor. In the fulminating cases tympany rapidly develops, constipation is absolute and vomiting persistent. The tenderness, at first local, becomes diffuse and is frequently most marked over the appendiceal region. We thus perceive what close resemblance it presents to intestinal obstruction and to appendicitis.

Fortunately the disease does not always pursue this rapid course, and the confusion is dispelled by the development of a tumor. The tumor of acute cholecystitis is usually symmetrical and smooth, tender on pressure, tense, non-fluctuating, and has the usual distinguishing marks of a tumor produced by a distended gall-bladder, as described under Gall-Stone.

In those cases in which there is virulent infection and peritoneal involvement, without the development of a palpable tumor, the symptoms are those of a peritonitis beginning on the right side, and a positive diagnosis of the true condition is often impossible.

DIFFERENTIAL DIAGNOSIS.—**Intestinal Obstruction**: The greatest confusion may arise from the resemblance of acute cholecystitis to intestinal obstruction. Pain, vomiting, constipation, tympany, and collapse are symptoms common to both. The temperature is the discriminating symptom. The occurrence of pyrexia of more than one or two degrees excludes intestinal obstruction prior to the advent of peritonitis. The development of a characteristic gall-bladder tumor would, of course, determine the matter.

Appendicitis: The common error in diagnosis is in mistaking an acute cholecystitis of the most fulminant type for appendicitis. The pain in appendicitis may be under the liver; the pain in cholecystitis may be in the right iliac region. The tenderness in cholecystitis may be elicited at McBurney's point. The general symptoms of bacterial invasion are common to both. Peritoneal phenomena are the same in both. And, indeed, the confusion grows more confounding the more carefully the symptoms are analyzed. Hotchkiss attaches great differential importance

to lateral compression of the lower ribs in developing pain as a symptom of cholecystitis. An intelligent history of the mode of onset is perhaps our best guide as to a diagnosis. If in either affection a tumor develops, discrimination should be made, but without it errors are possible and excusable.

Acute Pancreatitis: The history in both is very similar, the digestive disorders are common to both. Jaundice may be present or absent in both. The sudden violent onset of pain, though characteristic of pancreatitis, may be the initial symptom in cholecystitis. Acute pancreatitis gives pain in the epigastrium and to the left side, while cholecystitis is a right-side affection. Tenderness is deep-seated in pancreatitis, more superficial in cholecystitis. The tumor of pancreatitis lies under the stomach, is diffuse and ill-defined, often not palpable. If a tumor is present in cholecystitis, it occupies the region of the gall-bladder and is characteristic.

Perforated Gastric Ulcer: Perforation of a latent, unsuspected gastric or duodenal ulcer may appear in the guise of a perforative or gangrenous cholecystitis. The localizing symptoms would be our only guide as to the seat of the trouble.

PROGNOSIS.—One need only consider the pathology to recognize the gravity of acute phlegmonous or accidental cholecystitis. Under the paralysis of distention the cystic duct may relax and the contents of the distended bladder escape by natural channels. Should this not occur, the only other hope for spontaneous relief is adhesion and perforation into the intestine or through the abdominal parietes. The adhesions which quickly form in these cases offer a modicum of hope for localization and abscess-formation; but a more frequent and unfortunate termination is by way of septic peritonitis. The only case that the writer has observed developed a parotid abscess at the end of one week, and died a few days later of septic meningitis.

SURGICAL INDICATIONS.—*Cholecystectomy* is the indicated procedure. If gangrene, localized to the gall-bladder is present, it is certainly the only operation.

But a more conservative course may be pursued in the less virulent cases with patent cystic duct. *Cholecystostomy* with drainage, promptly done, with due precautions against peritoneal infection, offers a favorable operative prognosis.

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PRIMARY CARCINOMA OF THE GALL-BLADDER AND BILE-DUCTS.

DEFINITION.—An atypical neoplasm originating in the epithelial cells of the gall-bladder or extra-hepatic bile-ducts.

FREQUENCY.—In 1889 Ely was able to collect only 7 cases of indisputable primary carcinomata of the bile-ducts. Since that date contributions have been more numerous, necropsies have been made with greater precision, and it is now stated upon authority of statistics that primary carcinoma of the gall-bladder is found in 5 per cent. of all cases of carcinoma. Musser regards it not so rare a disease as we are led to believe. In 129 cases of new growths in internal organs tabulated by Norman Moore, 10 were cases of primary carcinoma of the gall-bladder and large bile-ducts. In 405 operations on gall-bladder and biliary passages by Mayo, 20 were for malignant disease.

ETIOLOGY.—**Age**: It occurs usually between the ages of fifty and sixty years.

Sex: Primary cancer of the gall-bladder is more frequent in women, while, be it remembered, primary cancer of the liver is more frequent in men. Courvoisier and Ames place the relative frequency of cancer of the gall-bladder in the sexes as 4 females to 1 male; Musser places it as 3 to 1. This disparity in sex is not so marked in primary growths of the large bile-ducts.

Gall-stones are often found in association with primary malignant disease of the extra-hepatic bile-channels. Courvoisier found calculi in 91 per cent., Musser in 92 per cent., and Ames rises to 95.4 per cent. Pathologists are naturally seeking an explanation of this remarkable association. Three views are possible: first, that calculi, acting persistently as local irritants, determine cancerous development; second, that the presence of the cancer begets such conditions as will favor the formation of gall-stones by biliary stasis; third, that the association is merely accidental. Brenner says that the occurrence of carcinoma in many cases without cholelithiasis divests that cause of its significance.

and he regards the association as a coincidence, a view that we think cannot be sustained. Gall-stones may have formed and escaped by the natural channels, or may have undergone disintegration; and in many cases the calculus is overlooked. In Warthin's case two calculi were found encysted in the tissues outside of the gall-bladder. Concerning the second theory that gall-stones are formed by the bile-stasis and changed conditions due to the new growth, clinical evidence altogether favors the conclusion that cholelithiasis exists prior to the development of the neoplasm. Furthermore, gall-stones are found in 95 per cent. of cases of primary cancer of the gall-bladder, while in 36 cases of secondary carcinoma of the liver and gall-bladder Beadles found calculi in not a single instance. The theory of irritation has, therefore, received general acceptance, and primary carcinoma of the bile-channels is commonly regarded as a sequel of gall-stones.

It appears to the writer that Roger Williams expresses the most tenable theory on this subject. He holds that the outbreak of cancer is invariably preceded by non-cancerous hyperplastic changes in the epithelia of the affected part. Such lesions are seldom limited to the precise starting point of the cancerous disease. This implies that the abnormal cellular activity, which at a given point culminates in cancer, affects also in a lesser degree the adjacent epithelia, so that there is a regional as well as a local predisposition. There can be no doubt that the morbid changes in the mucosa of the gall-bladder and biliary passages are the inevitable antecedents of calculus-formation. This view of the origin of the associate lesions, gall-stones and cancer, appears to be more in accord with the known facts than that which ascribes the origin of cancer to the irritation caused by the presence of the calculus. This would reduce the proximate causation of both gall-stone and cancer of the bile-channels to inflammation, the prime cause of which in turn may well be considered microörganismal. Though the etiological relation is somewhat obscure and difficult to define, yet clinical observation makes this salient fact conspicuous, that malignant degeneration is prone to develop in association with gall-stone, and but few instances are recorded of malignancy *developing after* the removal of the calculi. We are, therefore, fully warranted in placing carcinoma of

the bile-channels among the dangers and complications of cholelithiasis.

PATHOLOGY.—Microscopically, carcinoma of the biliary channels usually appears as a cylindrical-celled, medullary, or scirrhous epithelioma. Of these the columnar-celled adenocarcinoma is by far the most frequent. The origin of all forms is directly from the epithelium of the mucosa. Any part of the biliary tract may be the primary seat of the growth. Brenner says that carcinomata of the common duct show no predilection for any particular place. Courvoisier thinks that the neoplasm occurs most frequently in the fundus of the gall-bladder. Neoplasms of the common duct are small in size, rarely larger than a hen's egg, while those of the gall-bladder may attain to much larger dimensions. The scirrhous cancer is more limited in extent, and may appear as an annular, fibrous constriction about the duct, or, if of the gall-bladder, as an infiltrated, contracted mass. The columnar-celled variety yields a larger tumor with cyst-like spaces which may contain colloid material.

The tumor may be a smooth, flat mass, or nodular to the feel. The neoplasm extends as a polypoid in-growth, or as an infiltration along the ducts, leading to partial or complete occlusion of the lumina of the ducts. By continuity it may travel upward along the ramifications of the ducts to the liver. Secondary growths of the liver are rare. Lymphogenous metastases to the peritoneum, omentum, mesenteric and retroperitoneal glands are more in the ordinary. By contiguity the colon, duodenum, stomach, and pancreas may become involved. Warthin observed metastasis to the adrenals through the lymph-spaces of new inflammatory tissue.

In all reported cases involving the ducts there is stenosis, stasis of bile, and dilatation of biliary passages above the seat of obstruction. This results in enlargement of the liver, not infrequently in dilatation of the pancreatic duct, obstruction of the portal circulation, enlargement of the spleen, and ascites. Ulceration is a common complication; indeed, some authors maintain that it rarely takes place except in malignant conditions.

Neoplasms situated in the gall-bladder form palpable tumors; those primarily of the common duct rarely attain to sufficient size to be felt, and oftentimes previous inflammation has led to

contraction of the gall-bladder, preventing its distention. The tumor-mass is composed of several elements: the adherent omentum and intestines, the neoplasm itself, the fluid contents of the gall-bladder, and usually the contained calculi.

SYMPTOMS.—**Pain:** Pain is a variable symptom. Musser found it present in 62 per cent. of his cases. In some instances of common-duct obstruction it may be entirely absent. It may be expressed as sharp and lancinating, or as a dull ache or heaviness. Peritoneal inflammations and adhesions are important factors in its production. Movements of the body or muscular exertions excite pain. It is usually located in the right hypochondrium and radiates to the back. It is not distinctly paroxysmal or so severe as the pain of gall-stone.

Jaundice: In cancers primarily invading the gall-bladder or cystic duct jaundice is not necessarily an early symptom, but with extension to the hepatic or common ducts, or in primary growths of these channels, jaundice becomes marked; and when once it appears it is persistent, and progressively deepens. Painless jaundice, persisting, is especially significant of malignant obstruction.

Emaciation: This is an early symptom and may appear before there is pronounced jaundice; it may be attributed to the associate digestive disorder.

Digestive Disturbances: In the cases analyzed by Musser, he finds vomiting, constipation, and diarrhea common digestive disorders.

PHYSICAL SIGNS.—**Tumor:** A palpable tumor can usually be made out in all cases of carcinoma of the gall-bladder. It generally occupies the right hypochondrium, but may be felt in the umbilical or iliac regions. It varies from a flattened induration difficult to outline to a distinct mass as large as a grape-fruit. It is usually hard, irregular to the feel, tender upon pressure, and yields fremitus under palpation and auscultation. Areas of softening may be detected, due to cystic changes. It responds to the movements of respiration, but ordinarily is not freely movable by palpation.

In growths invading the common duct, with stasis of bile and distention of the gall-bladder, the tumor is somewhat elongated or pear-shaped, smooth upon its surface, tense or elastic to the

feel, may even be fluctuant. In cancer of the ducts without distention of the gall bladder it is difficult to detect a tumor.

Enlargement of the Liver: Hepatic enlargement is due to bile-stasis rather than to secondary growths, and hence is more constant in common-duct obstruction.

Ascites: While metastasis does occur, as we have shown in the pathology, Musser finds that it is not widespread, yet ascites is a common phenomenon in carcinoma of the bile-passages. It may be due to portal stagnation, or to peritoneal irritation from the primary growth or secondary deposits.

Enlargement of the Spleen: This is usually found in all carcinomata near the porta. It is mechanical in its pathogeny.

Metastases: Metastatic enlargement of other viscera and of mesenteric glands may be detected.

SUMMARY OF SYMPTOMS.—A general picture of carcinoma of the biliary passages may be thus depicted: There is anorexia, nausea, with attacks of vomiting, pain more or less acute in the region of the gall-bladder or deep in the epigastrium, more or less constant, with remissions and exacerbations, sometimes entirely absent at first; constipation, flatulency, and an occasional diarrhea; jaundice, at first slight, progressively deepening; marked emaciation and cachexia; in the majority of instances the development of a tumor, ascites, edema of the lower extremities; colorless, frequent, and involuntary stools; hemorrhages from the mucous membranes, petechiæ, stupor deepening into coma, and death.

DIFFERENTIAL DIAGNOSIS.—**Carcinoma of the Pylorus** and **Duodenum**: The gastric features are conspicuous; vomiting occurs with regularity some hours after eating; the vomitus may contain blood; there is consecutive dilatation of the stomach; jaundice is late except in cancer of the papilla duodenalis; enlargement of the liver is a late complication due to metastasis; the tumor is often to the left of the median line, more freely movable, less regular in outline, is never elastic or fluctuant, and yields an impaired tympanitic note upon percussion.

Carcinoma of the Pancreas: Emaciation, cachexia, and fatty diarrhea may be present without jaundice; the tumor is generally mid-costal in seat, fixed, and when of moderate size is reso-

nant upon percussion. With jaundice and enlargement of the liver, differentiation becomes very difficult.

Tumors of the Omentum and Colon: These are freely movable, irregular in outline, are not influenced by respiration, are not connected with the liver except by adhesions, yield a different note upon percussion, and have early and general metastasis. Jaundice does not appear until after hepatic secondaries occur.

Cancer of the Liver: Primary carcinoma of the liver is a very rare disease; secondary growths are usually multiple, cause general enlargement of the liver with distinct nodules; jaundice usually appears after the development of a tumor; and the parent growth may be detected.

Gall-Stone: The onset is sudden, pain severe and paroxysmal in character, often accompanied by chill and pyrexia; jaundice develops in two or three days, usually fades to recur with return of paroxysms; the tumor is elastic, smooth, globular or pyriform, freely movable, may fluctuate, frequently exists without jaundice, and may suddenly disappear.

PROGNOSIS.—The course of the disease is very rapid; patients frequently die within a few months, rarely live longer than a year. Musser places the average duration at 6⅔ months.

SURGICAL INDICATIONS.—If the disease is confined to the gall-bladder, and if there is no glandular enlargement or infiltration of the hepaticoduodenal ligament, *cholecystectomy* has been done, giving relief from symptoms and arrest of its progress for many months (?).

In cases of bile-stasis with distention of the gall-bladder, *cholecystostomy* will relieve the jaundice and its distressing phenomena.

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CONGENITAL OBLITERATION OF THE BILE-DUCTS.

There are some 60 cases of congenital obliteration of the bile-ducts recorded. The subject has received special study from Rolleston and Hayne, and from Thomson. The interest that it presents is largely physiological, in that it is shown that patients may live many months with complete occlusion of the bile-passages. In one notable case, operated upon by Mr. Treves, the patient was nineteen years old and had been jaundiced for sixteen years; it is doubtful if this case can be classed as congenital, since jaundice did not develop until the third year. Ford has collected 24 cases of cirrhosis of the liver which he believes were due to destruction of the common duct. Rolleston believes that a multilobular cirrhosis is a constant accompaniment of a congenital absence of the common duct. In seeking an explanation for this condition, says Rolleston, if we could determine that the changes in the bile-ducts antedated the cirrhosis of the liver, the relation of the two would be clearly understood. He offers the following hypothesis of the pathogeny: poisons pass by the blood from the placenta to the fetus through the umbilical vein; this toxic blood induces portal cirrhosis; the rest of the blood from the general circulation reaches the liver by the hepatic artery; the excretion of its toxic elements causes intrahepatic cholangeitis, so that in this way we have a mixed cirrhosis, and the inflammation extending to the larger ducts gives rise to a process analogous to obliterative appendicitis.

The main clinical features are as follows: the child is either jaundiced at birth or soon becomes so. The stools are colorless and the urine is bile-stained. The jaundice deepens, hemorrhages from the mucous membranes and the umbilicus may occur, liver and spleen progressively enlarge, and the child usually dies in two or three weeks, but may live for several months, finally dying of cholemia or some intercurrent trouble.

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PANCREATITIS.

DEFINITION.—A primary interstitial inflammation, with or without parenchymatous changes.

FREQUENCY.—In 1889, Fitz was able to collect 54 cases of acute pancreatitis. In 1898, Körte added 90 to this list. Since the latter date numerous cases have been reported, not because of increased frequency, but because of increased familiarity with the symptomatology. Thayer has seen 7 cases within six years. That it is a far more frequent condition than was supposed is now generally admitted. There were 7 cases reported in one year, 1897, to the London Medical Society.

ETIOLOGY.—**Age**: While somewhat more common between the ages of 25 and 50 years, it occurs at all periods of life. McPhedran reports a case in an infant nine months old. Several instances of its occurrence in advanced age are noted.

Sex: In 91 cases analyzed as to sex there were 59 males to 32 females.

In 1889, Fitz named, defined, and classified acute primary interstitial pancreatitis, a disease the effects of which have been recognized since 1641; the true etiology is only now in process of unraveling.

Neither the character of food nor the manner of eating, syphilis, or occupation can be regarded as predisposing causes. Alcoholism is classed as a predisposing or even exciting cause by Robson. The frequency with which pancreatitis occurs in obese patients, a large proportion of whom become rapidly fat, has led to the probably erroneous suggestion of a causal relationship. Rokitansky refers to a relation between parotiditis and pancreatitis. Inflammation may develop in the pancreas during the course of fevers, of typhoid especially. Infection of the pancreas from contiguous suppurative processes is not unusual. Its close relation to the stomach, duodenum, and bile-channels implicates it in perforative ulcerations of these organs. Fitz views it as a grave complication of the simple affection, gastroduodenitis.

The history of a few cases records an injury as a primary or

initiating cause. While hemorrhage, abscess, or gangrene may follow laceration of the structure of the pancreas, the vast majority of cases of pancreatitis develop without incident of injury.

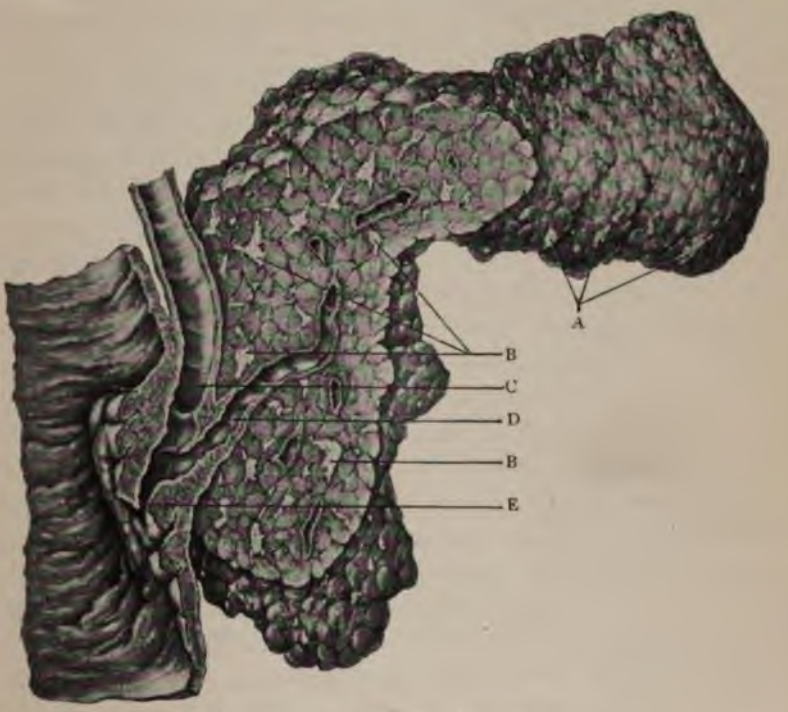
Gall-stones are active factors in the production of pancreatitis. The association of the two conditions is remarked by many writers, especially emphasized, however, by Barling, Robson, and Lund. The observations of Opie upon the effects of cholelithiasis have clarified the rather vague theories. Biliary calculi impacted in the common duct, producing ulceration and perforation, may excite pancreatitis. As an accompaniment of calculus of the common duct we find microörganisms and inflammation. It is not difficult to conceive that the infective process will invade the duct of Wirsung, and so introduced, the bacteria may initiate a pancreatitis. If the calculus is lodged near the orifice of the common duct, or if the concrement be very large, it may obstruct the pancreatic duct. Senn, in 1886, illustrated by experimentation the possibility of ligation of the pancreatic duct near the common duct as a surgical procedure. In all his experiments in which infection did not occur, parenchymatous degeneration and sclerosis of the gland followed. The atrophy could not be attributed to deficiency in the blood-supply, as it occurred regularly where this was not impaired. No allusion is made by Senn to the development of fat-necrosis, and he believes that pancreatic fluid is comparatively innoxious to the peritoneum. Opie in his work obtained somewhat different results; he found that ligation of both pancreatic ducts caused widespread fat-necrosis. His experiments show that when the pancreatic juice is obstructed in its outflow and dammed back upon the gland, it penetrates surrounding tissues and produces alterations, which serve as an indication of its presence as verily as jaundice indicates biliary obstruction. Katz and Winkler, in experiments upon 51 dogs, after ligating the pancreatic duct found extensive fat-necrosis; hemorrhage usually appeared in the necrotic areas, and there was leucocytosis and atrophy of the spleen. So far the resulting pancreatitis and fat-necrosis were attributed to the retained pancreatic secretion with due allowance for the element of infection. At an autopsy, following an unsuccessful operation for hemorrhagic pancreatitis, a minute calculus was found in the ampulla of Vater obstructing the duodenal orifice of the duct.

The pancreatic duct was stained with bile, and the parenchyma of the organ showed the presence of bile, though the hemorrhage somewhat concealed this appearance. Halsted thought that the bile had entered the pancreatic duct by retrojection from the common duct. Stimulated by this case, Opie performed 7 experiments with a view to demonstrating the effect of bile upon the pancreas. His conclusions are that necrosis of the parenchymatous cells and hemorrhage represent the primary action of the bile, and inflammatory reaction rapidly follows; and further, the frequent association of cholelithiasis with hemorrhage and gangrenous pancreatitis is the result of the impaction of gallstones at the duodenal orifice of the common duct, and of the penetration of bile into the pancreas. In only one experiment was the presence of bacteria demonstrated, although the experimental lesions were identical with those described as hemorrhagic and gangrenous pancreatitis in human beings. The value of the above observation cannot be overestimated. The proof is conclusive that sterile bile will cause pancreatitis. The conditions under which bile reaches the pancreas by retrojection are perhaps not limited to the complication of cholelithiasis—there are other causes of obstruction. Yet pancreatitis develops in the absence of all known causes, and post mortem search fails to demonstrate that it is consecutive to any other condition. The etiology of such cases rests in obscurity.

Mr. Robson asserts that the immediate cause of the various forms of pancreatitis is bacterial infection. Clinical observation and experimental work in a great measure sustain this position. The character of the process, which has been likened to diphtheria or to cancrum oris, is characteristic of a virulent infection. This infection may be hematogenous, or by extension from neighboring organs; but the most usual channel is by the route of the pancreatic duct. The microörganismal theory is further acceptable because it explains many of the clinical phenomena. Proof is not wanting of the potency of microörganisms to produce hemorrhagic and necrotic changes in the pancreas. The experiments of Flexner, Hlava, and others are affirmative; yet we know, also, that the same phenomena follow the introduction of irritants into the pancreatic duct or substance of the organ. The finding of microörganisms in the pancreas, in the fluid about

it, and in areas of fat-necrosis can scarcely be construed as establishing their causal relation. The organ in its relation to the colon and duodenum, if inflamed, would invite the penetration of bacteria, and it is surprising that they are ever absent; yet in numerous instances the most careful bacteriologic search has failed to demonstrate the presence of microörganisms after a fatal, destructive pancreatitis. In Halsted's case, previously referred to, the bacterial examination made by Bassett was practically negative. These notable exceptions justify us in accepting with some reservation the sweeping statement that pancreatitis is always an infectious process. Reasoning by analogy it is probably true, yet unproved.

PATHOLOGY.—On the threshold of the study of pancreatitis we are confronted with certain features that characterize the disease in its varied expressions. They are inflammation, hemorrhage, fat-necrosis, suppuration, and gangrene. Fitz asserted that interstitial inflammation was the essential feature of pancreatitis. Some effort is made to show that this inflammation is not primary, but reactionary in character. That there are instances of apoplexy or hemorrhage from the pancreas, not preceded by inflammatory changes, cannot be gainsaid. But that the ordinary hemorrhages which occur as a part of the phenomena of pancreatitis are attributable to an inflammation we think is clearly proved by the invariable finding of round-celled infiltration, an aggregation of leucocytes, and an increase of the interstitial connective tissue. Recurring to the experiments of Opie, we note this conclusion: "Necrosis of the parenchymatous cells and hemorrhage represent the primary action of the bile; an inflammatory reaction rapidly follows." There does not appear in the report of his experiments the evidence to sustain this conclusion, nor can I believe that he so holds, for he characterizes the hemorrhage as "hemorrhagic inflammation." It is notable that this term is used to describe the findings in the two animals that died within twenty-four hours; and the microscopical examination showed that the interlobular tissue was in many places much distended, containing red blood-corpuscles, polynuclear leucocytes, and fibrin. In those animals killed from the fifth to the seventh day fibrin was abundant in the necrotic interlobular tissue, and where destruction was not wide-spread there was active



PANCREATITIS WITH FAT-NECROSIS.

A. Areas of fat-necrosis upon the surface of the pancreas.
B. Areas of fat-necrosis within the parenchyma of the gland.
C. Ductus communis choledochus.
D. Ductus pancreaticus.
E. Papilla of Vater, opening of the ducts into the duodenum by a common aperture after junction just above.

This section makes very evident the ease with which the bile may and does enter the pancreatic duct when a stone becomes lodged in the papilla, below the junction of the common bile-duct and duct of Wirsung. Retrojection of bile into the duct of the pancreas is an important factor in the production of pancreatitis.

proliferation of interstitial tissue, replacing and destroying the parenchyma; polynuclear leucocytes were abundant, and the acini were separated by newly-formed interstitial tissue. Mr. Leith, in his pathological report of a specimen presented by Russell, makes this assertion: that the necrosis precedes the inflammatory reaction is clearly shown in some of the sections. I presume that this statement applies to the section and not to the entire pancreas. An inflammation confined to a portion of the pancreas may exert such pathologic influence as to cause necrosis of the organ remote from the area of inflammation, in which case sections of such necrotic spots would show absence of any inflammatory process. Flexner produced an inflammation of the pancreas associated with hemorrhage within forty-eight hours by injecting the bacillus pyocyaneus and the bacillus diphtheriæ into the pancreatic duct in animals. Inflammation is the legitimate consequence of infection; the accident of hemorrhage is probably explained by the intensity of the reaction and the peculiar texture of the pancreas. A hemorrhagic pancreas may be otherwise apparently normal until submitted to microscopical examination. Fitz attempted to show the relation that existed between pancreatitis and hemorrhage, but the meagerness of exact microscopical information precluded definite conclusions; nor does the author hold that the question is entirely disposed of.

The conclusion that one would reach is that bacterial infection is the essential and initiating factor, though not always demonstrable, and that inflammation follows. Hemorrhage may be one of its phenomena, and is generally so in the acute cases; in others, fatty degeneration and infiltration, with localized or multiple suppurations; or, there may be gangrene and sequestration of the entire gland, which destructive inflammation may or may not be accompanied by hemorrhage. In more chronic cases, especially in those accompanied by gall-stone, the inflammatory reaction is mild, the process is hyperplastic in nature, and the result is hypertrophy or induration of a part of the organ. We rest the question of the pathogenesis here.

Fitz gives us a pathological classification of pancreatitis which, perhaps, does not admit of clinical adaptation, yet denominates the most apparent pathology. *Hemorrhagic, suppurative,* and *gangrenous* are the varieties described by him; to these I would add *indurative pancreatitis.*

Hemorrhagic Pancreatitis: The presence of hemorrhage is usually suggested by some free, blood-stained peritoneal fluid; but in other cases nothing abnormal is found until the gland itself is reached. It may be dense, of normal size, and of firm consistency; or it may be much enlarged, soft and succulent. It usually appears discolored, of a dark reddish-brown or violet color; the discoloration may be localized, or general throughout, or in widely disseminated areas. Occasionally there is nothing unusual to be seen until section of the gland is made; we then find, usually in the interlobular spaces, hemorrhagic accumulations, which may show different ages. The duct and its ramifications contain blood or ichorous fluid. The hemorrhagic infiltration may extend into the parapancreatic tissue, into the mesentery, mesocolon, or fill the bursa omentalis. Gland-lobulation is often obscurely marked. Thromboses of splenic and portal veins are frequent. The spleen itself is usually small, though it may show no alteration whatsoever. In the retroperitoneal fat adjacent to the pancreas, upon the mesentery and omentum, small, round, opaque areas of disseminated fat-necrosis may be found. There is an acute cellular or fibrocellular infiltration of the connective tissue of the organ, with more or less extensive necrosis of the lobules. When the process is limited to a part of the gland, the boundary line between the diseased and uninvolved portions is marked by a zone composed of nuclear fragments, polynuclear leucocytes, red blood-corpuscles, and fibrin. Bacteriologic examination may be negative, or may show the presence of the colon-bacillus and other unnamed varieties.

Suppurative pancreatitis may appear as a single abscess involving any part of the gland; or the entire organ may be converted into an abscess cavity. In other instances there may be multiple foci of suppuration scattered throughout the organ. In these cases the pancreas appears swollen, dotted with numerous yellowish-white prominences arranged discretely or in groups; these granular areas contain granular corpuscles and detritus. There may be evidences of pancreatic hemorrhages, though suppurative pancreatitis is not necessarily preceded or accompanied by hemorrhages. The abscess-cavity contains a dirty green fluid, cheesy and necrotic material. The necrotic fat-material consists of opaque masses and granules, highly refracting globules and

needle-shaped crystals. The walls are ragged, and frequently the dilated ducts communicate directly with the pus-cavity. The parapancreatic fatty tissue forms a part of the wall and usually participates in the suppurative process. The pus may lie entirely retroperitoneally, dissecting between the folds of the mesocolon and downward to appear as a lumbar or iliac accumulation; more frequently, however, it communicates with the lesser peritoneal cavity, and on exposure the pancreas is found to lie in a puddle of pus. In Musser's case, in which there was a quart of pus, the confines were the stomach in front, the duodenum and transverse colon below, and the peritoneum above and behind. The pus had accumulated in the duodenojejunal fossa. He believed it a unique case, as it unquestionably was. In pancreatitis secondary to perforation of adjacent viscera, there is a fistulous communication. The spleen and liver are usually enlarged, thromboses of the splenic and portal veins are not unusual, causing obstructive ascites; pylephlebitis with multiple abscess of the liver appears as a fatal complication. Areas of fat-necrosis in the pancreas and parapancreatic tissues, and disseminated throughout the mesentery and omentum, are demonstrable in many cases, though not an invariable complication. Microörganisms are inconstantly found; usually the bacillus coli communis is present, or a large bacillus with square, clubbed end.

Gangrenous pancreatitis is usually the result of or is frequently associated with hemorrhage. It appears as a complication of perforating gastric and duodenal ulcers, and of gall-stones; and Fitz seems to think that the evidences of inflammation along the ducts are more demonstrable in this than in other varieties. Utilizing the records of Fitz, we find that the appearance of affairs in gangrenous pancreatitis varies somewhat with the duration of the disease. In the most acute cases the gland is increased in size, is dark red or mottled in color; sometimes it is transformed into a slaty-colored, stinking mass, the adjacent parts infiltrated with a purulent fluid, with some attempt at limitation by soft adhesions. In more advanced cases the pancreas is dark, dry, and firm. On section there are hemorrhagic masses with yellow spots of softening, obliteration of lobules with intervening opaque, white patches, like those in neighboring fat-tissue. In still more advanced cases the pancreas may lie free, as if

macerated, in a cavity containing ichorous fluid, which may communicate with stomach or intestines, or may burrow behind the colon to more dependent portions of the abdomen. Instances of sequestration of the pancreas and of its discharge by the bowel have been reported by Chiari. Venous thromboses are usually present, firm, and of a grayish-white color. There is an extension of the inflammatory process to the peritoneum. Splenic abscess is not uncommon. In more than one-half the cases of gangrenous pancreatitis there is such discoloration as to suggest hemorrhage. The fat-necrosis is disseminated from the pancreas and parapancreatic tissue to the peritoneum, mesentery, and omentum.

Indurative pancreatitis appears as a localized overgrowth of the connective tissue of the gland, with destruction of the parenchyma. Its inflammatory nature and association with obstruction from gall-stones have been demonstrated by Robson and by Barling. In Riedel's 3 cases the stony hardness of the tumor and its somewhat nodular or irregular surface led to the conclusion that it was carcinoma; but the autopsy showed interstitial pancreatitis. Fatty degeneration of the cells occurs, and here and there may be seen small white areas of necrosis.

Complicating Pathology.—**Fat-Necrosis**: A peculiar and interesting phenomenon which occurs in the course of acute pancreatic disease is the development in and about the gland and in widely disseminated areas throughout the abdomen of localized spots of fat-necrosis. The great accumulation of fat in these cases was attributed by Balser to a simple overgrowth of fatty tissue. Chiari regarded it as degenerative, merely a result of marasmus. Fitz considers it of inflammatory origin, while Rolleston attributes it to a trophic influence exerted upon the abdominal sympathetic system. Langerhans demonstrated that these necrotic areas consisted of the combination of fatty-acids with lime-salts. The fat-necrosis, according to his view, is due to the splitting up of the fat into fatty acids and glycerin; the latter is absorbed, but the acids, being insoluble, remain in the cells and unite with the calcium-salts, forming yellowish-white patches of various size in the subperitoneal fat, omentum, and mesentery. It cannot be confirmed that steapsin is the direct cause of the necrosis of tissue, but it is rendered probable because

of its constant presence in diseased areas, and by its absence from healthy fat. Senn long ago, and Flexner later, have shown that pure pancreatic juice is not harmful to and may be absorbed by the peritoneum; but when it escapes as the result of an inflammatory process the conditions are very different, and its deleterious influence, producing these areas of necrosis, is indicative of pancreatic inflammation. What part microörganisms may play in furthering the process Flexner thinks is doubtful. Welch does not think that fat-necrosis is attributable to primary colon-infection. Langerhans's view, while by no means confirmed, is acceptable; he believes that the primary lesion of the pancreas offers opportunity for the escape of the fat-splitting ferment from the organ into the surrounding tissue, resulting in secondary fat-necrosis. Fat-necrosis may be encountered unassociated with pancreatic lesion, but this is exceptional. It is impossible to define its relation to the various pathological varieties, yet we assert upon authority that it is frequently, though not invariably, associated with acute hemorrhagic pancreatitis, may not be observed in the suppurative type, but is usually present in gangrenous.

Symptoms.—Working upon the established basis that pancreatitis in all its forms is an inflammation, the symptoms do not conform to the incidence of hemorrhage, suppuration, and gangrene of the inflammatory process. It therefore appears unwise to attempt to adapt the symptomatology to the phases of pathology. We are sufficiently acquainted with this object to know that hemorrhagic, suppurative, and gangrenous pancreatitis all may pursue an acute course; that suppurative pancreatitis, as Mr. Fitz has clearly shown, shows a tendency to become subacute or chronic; and that indurative pancreatitis is necessarily chronic in its history, but may have an acute ending. Hence it follows that clinical ends are best subserved by treating the symptomatology of pancreatitis under the heads of *acute* and *chronic*.

Acute Pancreatitis: *History:* The history of digestive disorders, of gastroduodenitis, is so frequent in the anamnesis of acute pancreatitis that we should always accord it due consideration. Previous attacks of colic or a recognized history of cholelithiasis, especially of transient jaundice, have, in view of the latest studies upon this subject, assumed the greatest importance as an irri-

tating factor in the development of pancreatitis. Traumatisms cannot be ignored; in certain cases they are sufficient causes. It is, also, important that we ascertain if there has been a decided tendency to obesity of rapid progress. One-third of the cases give a history of alcoholic intemperance. In some few instances gross errors in diet are supposed to precipitate an attack. Suggestive as these facts in history are, yet in the majority of cases there is no ascertainable cause, no recognized prevailing previous condition. Acute pancreatitis often occurs in persons apparently in excellent health.

Mode of Onset: The onset is sudden, violent, and characterized by symptoms of individual importance.

Pain: Pain is the earliest and most emphatic symptom of acute pancreatitis. It is severe in all cases; Halsted describes it as frightful, more intolerable than the pain of gall-stone. Sometimes it is constant, but generally paroxysmal. It is usually felt in the epigastrium; later, it may be general to the abdomen.

Vomiting: Vomiting usually attends the pain, is copious at first, of a bilious character, later becoming less frequent and, perhaps, regurgitant, but never stercoraceous. The vomitus may be black; and it may show streaks of blood.

Hiccough: This is probably a reflex nervous symptom, distressing, and of frequent occurrence.

Constipation: Constipation is generally a pronounced condition; so conspicuous is it in the history of pancreatitis that it leads to the common error of diagnosing intestinal obstruction. In some cases diarrhea is met with.

Tenderness: With the acute onset, deep epigastric tenderness develops. This may not be detected upon light pressure; indeed, moderate pressure is voluntarily made by the patient; but firm, point-pressure elicits tenderness.

Shock or *Collapse:* Shock or collapse appears at variable times with relation to the onset of the symptoms. A patient may be seized with pain and vomiting, and may sink to the floor and rapidly pass into a condition of extreme collapse, dying in an hour or two. In other cases the shock may not supervene for several hours or days after the onset of symptoms. It usually portends an early exitus, as only a few patients have reacted from the condition of shock. The explanations given for this phe-

nomenon are not fully convincing; to ascribe the shock to hemorrhage is not tenable, for even in the so-called hemorrhagic pancreatitis the amount of the extravasated blood may be insignificant, and shock occurs with equal severity in pancreatitis without hemorrhage; it is attributed to interference with the functions of the great sympathetic ganglia, consequent vasomotor paralysis of the mesenteric vessels, and the draining into them of a large part of the blood, together with cardiac inhibition (Friedrich); another factor should be taken into consideration—bacterial poisoning. The constancy with which collapse appears in the clinical records of acute pancreatitis, the number of sudden deaths attributed to it, elevate it to a position of the greatest significance as a symptom, and I most emphatically do not lessen its importance, yet it must be recorded that cases are met with in which there is no shock. Halsted's case of acute hemorrhagic pancreatitis gave no evidence of shock; the patient's pulse was full and regular, beating 87, and he was walking about the room.

Pulse and *Temperature:* With the initial symptoms there is a frequent pulse; usually it progressively increases with the advance of the disease to become fast, feeble, and imperceptible in the stage of shock. If the patient survives the shock, or if the case pursues a subacute or chronic course, the pulse is characteristically weak and irregular.

In the ultra-acute cases the temperature is subnormal. Fever of slight elevation, perhaps a chill, may occur in other cases before the advent of shock. Suppurative cases show a septic chart.

Mania and *Delirium:* In the history of a few cases we note the occurrence of acute mania, a symptom which may be attributed to the intense suffering and auto-intoxication. Delirium or extreme mental lethargy is common in the course of suppurative pancreatitis. Wild delirium was present in the only case I have encountered.

Tympany: General abdominal tympany may sometimes occur. It is rather a symptom then of general peritonitis; but localized distention of the epigastrium, resonant upon percussion, is an early phenomenon. The patient expresses a sense of fullness and distention, which is, perhaps, as significant as the physical sign.

Ascites: In the most acute cases free fluid may occasionally be detected in the abdomen; this may be blood, but is usually sero-

hemorrhagic. In cases less acute, for instance, in Musser's with a duration of three weeks, there was great accumulation of fluid which required tapping; it reformed in 18 hours. The occurrence of portal and other venous thrombosis explains the ascites and edema.

Jaundice: Jaundice occurs in a limited number of cases. It may exist before the onset of distinct pancreatic symptoms. Cholemia should certainly be regarded as a serious complication predisposing to hemorrhage.

Tumor: A tumor can rarely be detected in the fulminant cases of pancreatitis. There is a resistance but not a defined tumor. The epigastric swelling has been exceptionally noted to be pulsatile, sometimes causing a splashing sound in the stomach; and a murmur may be heard over it (Trollope). In the suppurative form, in which the disease pursues a less acute course, a deep-lying tumor may be felt above the umbilicus. Palpation is made difficult because of the epigastric distention or ascitic fluid, and great tenderness.

Urine: Glycosuria, maltosuria, indicanuria, etc., have been investigated in expectation of being proved of diagnostic importance, but they are not present with sufficient regularity, though glycosuria, when it is present, is of both diagnostic and prognostic value.

Mr. P. J. Cammidge has developed a test for the glycerine which should be found in the urine as the result of fat necrosis, and this so-called "pancreatic reaction" is of undoubted value, though it has not yet been used to any extent except by the originator. The test is made as follows:

A specimen of the twenty-four hours' urine, or of the mixed morning and evening secretions, is filtered several times through the same filter paper and examined for albumen, sugar, bile, urobilin and indican. A quantitative estimation of the chlorids, phosphates and urea is also made, and the centrifugal deposit from the urine examined microscopically for calcium oxalate crystals. If the urine is found to be free from albumin and sugar, and of an acid reaction, 1 c.c. of strong hydrochloric acid (specific gravity 1.16) is mixed with 20 c.c. of the clear filtrate, and the mixture gently boiled on the sand-bath in a small flask, having a long-stemmed funnel in the neck to act as a condenser. After ten minutes' boiling the flask is well cooled in a stream of water, and the

contents made up to 20 c.c. with cold distilled water. The excess of acid present is neutralized by slowly adding 4 grams of lead carbonate. After standing for a few minutes the flask is again cooled in running water and the contents filtered through a well-moistened, close-grained filter paper until a perfectly clear filtrate is secured. The filtrate is then well shaken with four grams of powdered tribasic lead acetate and the resulting precipitate removed by filtration, an absolutely clear filtrate being obtained by repeating the filtration several times if necessary. The large amount of lead in solution is removed either by treatment with a stream of sulphuretted hydrogen or by precipitating the lead as a sulphate. For this purpose the clear filtrate is well shaken with two grams of finely powdered sodium sulphate, the mixture heated to the boiling point, then cooled to as low a temperature as possible in a stream of cold water, and the white precipitate removed by careful filtration; 10 c.c. of the perfectly clear transparent filtrate is made up to 18 c.c. with distilled water added to 0.8 gram of phrenylhydrazin hydrochlorate, 2 grams of powdered sodium acetate and 1 c.c. of 50 per cent. acetic acid contained in a small flask fitted with a funnel condenser. The mixture is boiled on a sand-bath for ten minutes, and then filtered hot through a filter paper moistened with hot water into a test-tube provided with a 15 c.c. mark. Should the filtrate fail to reach the mark, it is made up to 15 c.c. with hot distilled water. In well-marked cases of pancreatic inflammation a light yellow, flocculent precipitate should form in a few hours, but it may be necessary to leave the preparation to stand over night before a deposit occurs. Under the microscope the precipitate is seen to consist of long, light yellow, flexible, hair-like crystals, arranged in sheaves which, when irrigated with 33 per cent. sulphuric acid, melt away and disappear in from ten to fifteen seconds after the acid first touches them. The urine employed for the experiment should be fresh, and not have undergone fermentative changes. If alkaline in reaction, it should be made acid with hydrochloric acid before the test is commenced; any glucose that may be present should be removed by fermentation after the urine has been boiled with the acid, and the excess neutralized.

Stools: The condition of constipation which prevails in the acute cases precludes a satisfactory examination of the stools. In those cases that pursue a more protracted course, when diarrhea super-

venes, the stools contain fat-droplets. Occasionally there is melena. Gangrenous portions of pancreatic tissue may be passed by the bowel when there is an intestinal communication with the abscess cavity. More important is the appearance of unstriped muscular fibers in the dejecta, evidence of lack of pancreatic secretion.

Chronic Pancreatitis: The symptoms pursue a chronic course in two forms of pancreatitis, *suppurative* and *indurative*. Suppurative pancreatitis has an acute onset which may lead to an early termination in collapse and death. Gradually the acute symptoms may subside. The further progress of the case is marked by progressive and rapid emaciation, a fluctuating or septic temperature with intercurrent chills, profuse, watery alvine discharges, which may be much discolored or decolorized, containing fat-globules. An epigastric tumor may be felt. Lumbar and iliac accumulations of pus develop. Anasarca and ascites become apparent. The liver may enlarge; Fitz found the spleen usually small. Death finally occurs from exhaustion and marasmus.

We are especially indebted to Mr. Mayo Robson for the symptomatology and treatment of localized hypertrophic cirrhosis of the pancreas or indurative pancreatitis. The symptoms presented by this condition are those of common-duct obstruction, partial or absolute. He specially calls attention to decolorized stools in some cases in which there is no jaundice, citing Walker, of Peterborough, as authority that, even though bile was present in the intestinal canal, the absence of pancreatic secretion would lead to pale-colored stools. Glycosuria, lipuria, and fat in the stools too seldom occur to be of much use in diagnosis, but when present are of great diagnostic value. The Cammidge test is of special value here and the combination of the urinary pancreatic reaction with fat and undigested muscle in the stools is considered absolutely diagnostic by Robson. Usually these cases of indurative pancreatitis are associated with calculus in the common duct, and, therefore, give a history of biliary colic, and not infrequently of the intermittent fever. Other cases, however, show painless obstruction with the development of a tumor, and such cases present the clinical features of malignancy. Especially striking is the rapid and marked emaciation in these chronic cases of pancreatic disease, which Hemmeter attributes to the exclusion of pancreatic secretion.

DIFFERENTIAL DIAGNOSIS.—The clinical picture of acute pancreatitis is stereotyped; sudden onset, violent epigastric pain, distressful vomiting, perhaps fever, then collapse and death within a few days. Further analysis of the symptoms shows that we may group cases under several heads: (1) those few cases attributable to traumatism, in which the classical symptoms follow with moderate rapidity; (2) those cases with a history of cholelithiasis—here the symptoms develop with the phenomena of the dual affection, and are most confusing in differentiation; (3) those cases with an acute onset, but in which a tumor, occupying the region of the pancreas, develops. In all such cases there are the symptoms of a peritonitis starting in the epigastric region; yet it must not be inferred that every case of pancreatitis offers so well-characterized features. We have noted the difficulty that Halsted encountered in diagnosis; all the symptoms save pain were wanting, and "the only sign which this obscure case presented was the white print of finger tips in a slightly cyanosed field just over the site of greatest pain." As clearly drawn as Mr. Fitz has given us the symptomatology of this disease, the correct diagnosis is rarely made, and we believe that to Thayer and Finney still belongs the credit of having had the only case of correct diagnosis confirmed by operation and rewarded with recovery.

From **intestinal obstruction** pancreatitis may be distinguished by the seat of pain and tenderness, an unusual site of obstruction, by the absence of general tympany, by the occurrence of fever, by the absence of stercoral vomit, and by the possibility of obtaining by enemata fecal discharges or free passage of gas.

Visceral Perforation: The symptoms of acute peritonitis originating in the upper zone of the abdomen would suggest gastric or duodenal perforation. The previous history would here be our chief guide to differentiation. We would be further aided in recognizing perforation by the presence of general tympany, indicative of the presence of free gas in the peritoneal cavity. After perforation of the stomach or duodenum vomiting usually subsides until the advent of peritonitis; this is not so in pancreatitis. In pancreatitis the tenderness is deep-seated; while in perforation-peritonitis it is superficial, and there is hyperesthesia. If collapse is profound in pancreatitis, death is likely to follow;

in perforation of viscera the immediate shock is followed by reaction.

In **irritant poisoning** the diagnosis is made by the history of the case, and by the chemical analysis of the vomited material. Examination of the buccal cavity in certain forms of poisoning may show the effects of the irritant.

Gall-stone: Especial difficulty is to be encountered in those cases of biliary colic not associated with jaundice. But here the repose between the paroxysms, the prompt reaction, the local tenderness over the region of the gall-bladder, the referred pain to the back and shoulder, sometimes the development of general tympany rather than the localized epigastric distention, and the history of previous attacks, guide us to the correct localization of the symptoms.

PROGNOSIS.—Acute pancreatitis is a very fatal disease. Death may occur at once from collapse, usually within four or five days from septic peritonitis. The suppurative form may be protracted through several weeks. It is possible for recovery to take place by the establishment of a fistulous communication with the intestine and the discharge of the contents of the abscess or of the gangrenous and sequestrated pancreas through that channel. The only case of suppurative pancreatitis recognized and operatively treated by the author was in a man aet. 46, a patient of Dr. J. R. Banks, of Nashville. The abscess was in apposition with the parietes just above the umbilicus. About one pint of pus was evacuated, and the cavity packed with gauze; the edges of the wound became gangrenous, finally involving an area as large as a dinner plate. The patient died on the nineteenth day after operation—the forty-second of his illness.

TREATMENT.—**Acute Pancreatitis:** This condition demands immediate abdominal section with incision and drainage of the pancreas. Cases that live beyond the acute stage and are operated later are more apt to recover than those subjected to immediate section, but this does not justify the delay. It simply amounts to waiting for selective action on the part of Nature, which procedure helps surgical statistics, but does small credit to the judgment and courage of the surgeon.

The pancreas may be approached transperitoneally through an anterior incision, preferably in the median line above the umbilicus.

After entering the greater cavity of the peritoneum, it is best in most instances to open the lesser cavity through the gastrocolic omentum, though at times it may be desirable to go through the gastrohepatic omentum or the transverse mesocolon. The head of the pancreas can be reached by incising the peritoneum to the right of the duodenum, and turning that organ to the left, while the tail of the organ is accessible above the transverse colon to the left of the stomach.

The pancreas can be reached retroperitoneally by an incision in either lumbar region. By this method of approach we get access to the head or tail alone, and for this reason it is little used except for drainage, and then only after that organ has been thoroughly explored through an anterior incision.

Pancreatic abscess requires incision and drainage. So far as drainage is concerned the posterior incision is best, but the abscess may not be accessible from this direction, and usually an anterior incision will be required for investigation, as is already stated.

Chronic indurated pancreatitis requires drainage of the bile channels after any obstruction present had been removed. Drainage of the gall bladder either externally or into the bowel will at times suffice, but usually incision of the common duct at the ampulla, either transduodenal or retroduodenal, will be necessary for the removal of biliary or pancreatic calculi.

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CANCER OF THE PANCREAS.

DEFINITION.—The term is limited to a primary carcinomatous involvement of the pancreas. Lymphoma and sarcoma are other forms of malignant tumor very occasionally met with; and as they present the same symptoms, they require no special consideration.

FREQUENCY.—Carcinoma has been considered the most frequent disease of the pancreas; this view will probably not obtain now that we understand the nature of obstructive pancreatic cirrhosis. In 3,233 autopsies Flavio found only two cases of carcinoma of the pancreas. At the general hospital in Vienna, Biach found 22 cases of cancer of the pancreas in 18,069 autopsies. Richardson, of Boston, encountered cancer of the pancreas 5 times in 23 fatal gall-bladder obstructions. Mr. Robson has seen 50 cases.

ETIOLOGY.—**Age:** Cancer of the pancreas is rarely met with before forty years of age.

Sex: DaCosta found it more common in males than in females. In 50 cases analyzed by Baldwin, there were 33 males and 17 females.

It is not known whether diet, injuries, or previous inflammation have any influence upon the development of cancer; but the association of cholelithiasis with cancer of the pancreas occurs sufficiently often for us to attach some etiological significance to the irritating effects of the calculi.

PATHOLOGY.—Carcinoma of the pancreas is usually of the hard or scirrhous variety, though the medullary and colloid are occasionally found. Primary carcinoma rarely appears as a disseminated affection throughout the gland. The head of the pancreas is the usual seat; or it may spring from either the body or tail. Its origin is from the epithelial cells lining the ducts, and from the acinous cells. Metastasis to the lymphatic glands, liver, and spleen occur. The stomach, duodenum, colon, and peritoneum may be directly infected. By pressure the duodenum or pylorus may become obstructed, leading to gastrectasia (Taliaferro).

By pressure upon the vena cava, edema of the lower extremities develops as a frequent symptom. Extraneous or thrombotic occlusion of the portal vein gives rise to ascites. Chylous ascites, which Flavio regards as a pathognomonic symptom, is not necessarily due to rupture of the thoracic duct, it may occur from exosmosis. Leo has seen hydronephrosis from pressure on the ureter. The most frequent and confusing pathological complication is obstruction of the common duct, with jaundice and dilatation of the biliary channels. The liver, unless secondarily involved, is not necessarily enlarged.

Symptoms.—Loss of appetite, great muscular weakness, extreme prostration, and a deep-seated pain in the region of the epigastrium, which may be acute and paroxysmal, or a dull, constant ache, are early symptoms in a person over forty years of age which should suggest cancer of the pancreas. If jaundice occurs in the beginning of the illness, with sugar in the urine and fat in the feces, they have considerable supposititious force. Glycosuria as a symptom of pancreatic disease, especially of cancer of the pancreas, at one time was highly valued. Bard and Pic find that of 150 cases of carcinoma of the pancreas recorded in literature, in only 17 was there glycosuria; they claim that in the later stage of the disease the cancer-cells have the histological character of pancreatic cells, and also their physiological function of producing glycolytic ferment, which is diminished in the early stage because of pressure and sclerosis; the restoration of the glycolytic ferment leads to a normal transformation and consequent disappearance of glycosuria—an explanation very fitting, no doubt, to some cases; but there are many cases of cancer of the pancreas in which glycosuria is never present. Decolorized stools may be discharged even in cases in which there is no jaundice. A fatty diarrhea is only an occasional symptom. The pronounced cachexia, great weakness, and local tenderness may in some instances be the only symptoms. The digestive disturbances, nausea and vomiting, fatty diarrhea, and glycosuria may all be absent. Great nervous prostration and mental lethargy, sometimes delirium, even acute mania, occur in the course of cancer of the pancreas. In those cases examined the blood showed leucocytosis, blood-plaques, and decrease in hemoglobin. Usually jaundice appears, especially if the cancer involves the head

of the pancreas. Painless jaundice with distention of the gall-bladder indicates malignancy, probably of the pancreas. Ascites is a late and constant symptom; its chylous character is mentioned by Flavio and Vance. Edema of the extremities may occur independently of an abdominal effusion. In those cases in which jaundice is absent, the skin assumes an ashen or silvery-gray hue; petechiæ may appear. In the early history of Trower's case bullæ appeared upon the dorsal surfaces of both hands, a variety of pemphigus which is not unusual in wasting diseases of the liver, spleen, and pancreas.

The tumor may remain small and not easily detectable, if it involves only the head of the pancreas, since it is concealed by the liver. In some instances it attains to considerable size; in Ruggi's case it measured 25 x 13 cm. When of large size the tumor occupies the umbilical rather than the epigastric region, underlies the stomach, and is, therefore, tympanitic upon part of its surface; the colon is generally pushed downward, and can be outlined by percussion after inflation along its lower border.

DIFFERENTIAL DIAGNOSIS.—**Carcinoma of the Pylorus:** The celiac neuralgia, the great emaciation and cachexia indicate the malignant character of the affection. It is often very difficult, even impossible, to define its origin. It is frequently confounded with cancer of the pylorus from which it may be distinguished by the history of the case, the conspicuousness of the stomach-symptoms, the absence of hydrochloric acid in the stomach-contents, the constant association of gastric dilatation with pyloric disease; and lastly and chiefly, we rely upon the physical signs obtained in the examination of the tumor: a pyloric tumor is more superficial, often to the right of the median line, in some instances easily displaced to the left, may be dull upon light percussion, but the deep stroke brings out the underlying resonance, just the reverse of what would be obtained in a pancreatic tumor.

Indurative pancreatitis, especially when associated with obstruction of the common duct by calculus, may present all the symptoms of malignancy. The history of biliary colic, the early occurrence of jaundice, and the absence of distention of the gall-bladder would point to the inflammatory rather than to the malignant character of the tumor. Yet even after the abdomen is opened, digital examination showing a hard, nodular tumor may

lead us to the erroneous diagnosis of its malignant nature. Recovery which follows cholecystostomy and drainage shows its inflammatory nature. Richardson, and Robson warn us against this error.

Abdominal Aneurism: The wasted condition of the patient, his pronounced cachexia and great weakness, and probably fatty diarrhea and glycosuria, are symptoms not present in abdominal aneurism in a stage requiring differentiation. Ascites and jaundice, symptoms so common in cancer, are not present in the early stages of aneurism. Our chief reliance, however, lies in the examination of the tumor itself, which, when aneurismal in character, shows the characteristic expansile pulsation.

PROGNOSIS.—The course of the disease is progressively downward, and the prognosis is absolutely hopeless. Five months after supposed complete extirpation of the pancreas for a large carcinoma, Ruggi's patient was in perfect health.

CONTRAINDICATIONS TO OPERATION.—The usual location of the disease in the head of the pancreas, the deep-seated situation of the tumor, the frequent involvement of surrounding structures are the difficulties which preclude excision, and Mayo Robson doubts if it is ever justifiable.

SURGICAL INDICATIONS.—In those cases of pancreatic tumor attended by the symptoms of biliary obstruction, the indication is to explore. The happy results that follow *cholecystostomy* and drainage justify this, at least, as a palliative measure; we may be rewarded with an absolute cure when the tumor is only inflammatory.

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PANCREATIC CALCULI.

Calculi, evidently of biliary origin, are occasionally found in the pancreatic duct. These must be distinguished from those concrements that are formed by precipitation of the salts from the pancreatic juice, uniting with organic elements, the product of inflammation, resulting in agglutination and the formation of a stone. Calculi are frequently found in connection with cysts of the pancreas, and it is supposed that by obstructing the duct they give rise to dilatation and cystic formation. It is more probable that the concrements are secondary to pancreatic inflammation and stagnation of secretion. Shattuck does not believe that these concrements, composed chiefly of calcium oxalate, can be of bacterial origin, but thinks them hematogenously derived. It has also been observed that biliary, renal, and pancreatic calculi may coexist in the same individual; such a lithiasis is attributed to arthritis uratica.

The concrements consist usually of calcium carbonate and phosphate. In size they vary from small sand-grains to as large as a walnut. They may be very numerous, but are generally single. The presence of calculi in the pancreas is usually associated with inflammatory changes in the substance of the gland; it is difficult to determine whether these changes are primary or secondary. The inflammation may be acute, resulting in abscess-formation, or chronic, indurative in character. The gland may possibly become enlarged in some instances, but in the great majority of cases atrophy occurs, the acini disappearing, and the pancreas becoming small and hard, and often flattened out. The bile-duct may be obstructed as a result of this cirrhotic change, or by direct pressure of the concretions.

The symptoms attending the presence of pancreatic calculi are such as are observed in other diseases of that organ, and closely resemble biliary colic. The paroxysms of pain, vomiting, and occasional transient jaundice are inconclusive. However, when these phenomena are coupled with those digestive disturbances

indicating an absence of the pancreatic ferment, the evidence becomes more direct against the pancreas as the organ at fault. In 70 cases of lithiasis Oser observed that diabetes was recorded 24 times. Lancereaux says that transient diabetes occurs with pancreatic colic. Kinnicut can find only six cases recorded in literature in which a positive diagnosis of pancreatic lithiasis was made, to which he adds one of his own. But clinical observations have not been sufficiently frequent or accurate to enable us to assign a specific symptomatology to pancreatic calculi.

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CYSTS AND PSEUDOCYSTS OF THE PANCREAS.

DEFINITION.—A *true cyst* of the pancreas is a sacculated accumulation of fluid originating in the pancreas and retaining an anatomical connection with the organ. *Pseudocysts* are accumulations of fluid in the lesser peritoneal cavity about the pancreas.

FREQUENCY.—In the records of 6,000 post-mortem examinations at Guy's Hospital, Malcolm found mention of four pancreatic cysts. Körte, in 1898, collected 121 cases subjected to operation by various surgeons; Bessel-Hagen, in 1900, increases this number to 149.

ETIOLOGY.—**Age:** Railton reports a case in an infant six months old. It is most usually met with in middle life.

Sex: Cysts of traumatic origin occur more frequently in males; when due to other causes they occur with equal frequency in the sexes.

The experiments of Senn demonstrated that ligation of the pancreatic duct under aseptic technic produced atrophy of the gland, and never cystic dilatation of the duct. This observation disturbed somewhat the accepted pathogenesis of pancreatic cysts. Virchow, considering the origin of a cyst of the pancreas as analogous to *ranula buccalis,* designated it '*ranula pancreatica,*' a retention-cyst. That general ectasia of the pancreatic ducts or a sacciform expansion may take place after obstruction, associated with or due to inflammation or calculi, seems beyond dispute; the ligation of a healthy duct in no way disproves this. The parenchymatous alterations consequent upon interstitial inflammation, attributable to infection through the duct of Wirsung, have been fully discussed under "Pancreatitis"; and that these changes may act as subsidiary causes to cystic formation is especially well shown by those cases of multiple cysts of the pancreas described by Klebs as '*acne pancreatica.*' Any obstruction of the pancreatic duct with inflammation may then be regarded as a cause of retention cysts.

Proliferating cysts or cystadenomata are true neoplasms of unknown origin.

Traumatism appears so conspicuously throughout the records of cysts of the pancreas that it must be regarded as the most generally active cause. An injury, resulting in bruising or laceration of the pancreatic structure with hemorrhagic extravasation, may be followed by degeneration and liquefaction of a certain amount of the involved pancreatic tissue with secondary formation of a cyst, its walls being composed of newly-formed connective tissue.

Jordan Lloyd does not regard traumatic cysts as true pancreatic cysts, but as effusions into the lesser peritoneal cavity, or *pseudocysts*.

PATHOLOGY.—True cysts of the pancreas are retroperitoneal tumors; pseudocysts are intraperitoneal accumulations of fluid. To reach a true pancreatic cyst from in front four layers of the peritoneum are necessarily divided. The anterior and lateral walls of a pancreatic cyst of moderate size are in relation with the posterior or parietal layer of the lesser peritoneum, separated from it by only a very thin layer of connective tissue, while the under surface of the tumor's relation depends upon its size, and its direction of evolution.

True cysts of one variety develop by dilatation of the duct, as is conclusively proved in some few instances by the finding of the duct's distal or proximal end in communication with the cyst's cavity, and furthermore, by the showing microscopically of a cylindrical epithelial lining. It is obvious, however, that altered pancreatic secretion and inflammation may destroy the epithelial lining and obliterate the duct; hence the absence of these anatomical tests does not disprove the duct-origin of the cyst. Such tumors are usually single, but may be multiple, giving the gland the appearance of a bunch of grapes. They may spring from any part of the canal. They are slow of evolution, increase in size by coalescence of adjacent cysts or by hemorrhages into the preformed cyst-cavity. Fitz, in August, 1900, was able to collect from the literature 11 cases of multilocular pancreatic cysts. Papillomatous excrescences are sometimes found on these cysts, and the comparison between them and proliferating ovarian cystomata has often been made.

A second variety, due to inflammation or traumatism, and usually seated in the tail of the pancreas, develops from hemorrhagic

effusion into the glandular tissue, which results in degenerative changes, leading to the formation of an adventitious wall of inflammatory material and atrophied pancreatic tissue, into which hemorrhages repeatedly occur. Such tumors are distinguished by their dark color, hemorrhagic contents, rapid growth, and anatomical connection with the organ. The sac-wall consists of connective tissue of greater or lesser thickness; in Tilger's case the wall of the cyst was calcified in places, but this is exceptional, as the growth is usually very rapid and the wall quite thin. Friedrich describes these as apoplectic cysts, attributing their origin to hemorrhagic foci in the pancreas due to preëxisting changes in the vessels, in short, to inflammation. Between 1880 and 1894, Tilger collected 47 cases of this variety of blood-cysts, all connected with the tail of the pancreas.

Contents: The contents of pancreatic cysts are usually fluid, brownish-red or dark-green, sometimes colorless, alkaline in reaction, and containing albumin, fat-globules, crystals of cholesterin, white and red corpuscles, and inorganic salts—that is, the contents are sanguinolent in most cases, though exceptions are met with. Of the specific ferments the diastatic is the most common one found in the cystic fluid; the fluid may also possess emulsifying properties; tryptic ferment may occasionally be found. Pure pancreatic secretion may be innocent, but the fluid from the cyst quickly excites irritation.

The size of pancreatic cysts varies from a small vesicle to an immense sac containing four gallons.

Jordan Lloyd does not believe that the fluid accumulations following injury to the pancreas are cystic in character, but that they are an admixture of blood and pancreatic secretion in the cavity of the lesser peritoneum. Following a slight injury of the pancreas, a small hemorrhagic effusion may be absorbed without sequence; but if there is free hemorrhage, and especially if the duct of the pancreas is opened, there follows in a short time an accumulation of fluid about the pancreas. The delay sometimes observed between the incident of injury and the development of symptoms may be explained by the progress of the degenerative changes. Careful analyses of cases by Mr. Lloyd show that these effusions are of the same chemical composition as true pancreatic cysts, that they yield the same symptoms and

physical signs, and that surgeons base the diagnosis of the pancreatic origin of a cyst upon the character of the contents rather than upon an actual demonstration of its anatomical connection; that the mere finding of the pancreas by an examining finger from within an intra-abdominal sac is no proof that that sac is adventitious and of pancreatic origin.

Complicating Pathology.—Pancreatic cysts by their size, direction of growth, and relations have important effects upon neighboring organs, displacing and compressing them. We find the heart pushed upwards, the pylorus and duodenum obstructed, with consequent gastric dilatation, the intestines occluded, the ureters compressed, causing hydronephrosis; the solar plexus and celiac ganglia pressed upon and involved in adhesions. Salzer refers to one instance in which the cyst developed behind the abdominal aorta and vena cava, pushing these vessels forward toward the anterior abdominal wall, where the pulsations of aorta were plainly visible. The splenic artery, owing to its close anatomical relation to the tail of the pancreas, has been found to lie across the anterior surface of the cyst. The common occurrence of hemorrhage into the cyst results in its sudden distention, and sometimes rupture, under which circumstances the patient may die from acute anemia or peritonitis. The cyst-wall may undergo degenerative changes from pressure-necrosis, permitting an escape of its contents into the peritoneal cavity, or, by fistulous communication, into the alimentary canal. Tumors diagnosed as cysts of the pancreas have been known to disappear; it is conceivable that a hemorrhagic effusion might so behave. Observations are not sufficiently accurate in warranting us to believe that a true cyst of appreciable dimensions could rupture without marked peritoneal irritation.

Symptoms.—The symptoms of pancreatic cyst may appear only with the detection of the tumor, but usually there are antecedent symptoms which possess an etiological bearing. In no inconsiderable number of cases there is a history, more or less definite, of an abdominal injury. In many cases the association is very close and the patient connects his symptoms with the injury; in others, years have elapsed. The traumatic force is generally a squeeze or pressure of a heavy weight rather than a sudden blow. In other instances the development of the cyst

follows typhoid fever, attacks of colic, or gastroduodenal disorders. It must be understood that the symptomatic course of pancreatic cysts is comparatively acute; a few weeks may cover the entire history. But there are instances in which the cyst has been known to be present for years, the accidents of hemorrhage, rupture, or intestinal obstruction bringing its course to a sudden termination. In one of my cases five years had elapsed since the causative injury; the patient had suffered great discomfort continuously, but a tumor had appeared only three months before operation. Its rapid development was probably due to hemorrhage.

The development of a pancreatic cyst initiates very variable and indefinite subjective symptoms. They appear usually in the following order:

Pain: Pain may develop suddenly, or there may be a history of epigastric pain for years. It is usually constant, described as a heaviness or weight, but may be sharp and paroxysmal in character, striking through to the back, and irradiating over the abdomen. This celiac neuralgia is not so intense as in malignant neoplasms, yet in exceptional instances it produces collapse of grave severity.

Digestive Disturbances: Perversions of appetite, nausea, and vomiting are common attendant symptoms, attributable, perhaps, chiefly to pressure. The gastric distress is relieved by free emesis or belching. The compression of the pylorus or duodenum, with consequent dilatation of the stomach, and characteristic and copious vomiting, is a misleading feature. Diarrhea or constipation may prevail; the stools possess but little diagnostic significance. Bloody stools may be intercurrent phenomena in the history of pancreatic cysts. Undigested unstriped muscular fibers may be found in the bowel-motions. It may be said that neither sialorrhea, steorrhea, jaundice, nor glycosuria are characteristic symptoms; taken either separately or collectively, they are merely suggestive but not diagnostic of pancreatic disease. Emaciation is progressive during the course of pancreatic cysts. It is attributable to disturbance of the functions of viscera from direct pressure, to involvement of the solar plexus, and to the absence of the pancreatic ferments from the alimentary canal.

Dyspnea and palpitation are sometimes distressing symptoms when the stomach is full. The patient breathes freely only when he is in a sitting or half-reclining position. The pulse is usually feeble and fast. There is no elevation of temperature unless complications exist. Mental disturbances, melancholia especially, are emphasized as a common symptom by Doran.

PHYSICAL SIGNS.—**Inspection:** The great emaciation, the discolored, dry skin, the jaundiced tinge, the anemic mucous membranes, the profound hopelessness or depression, as depicted in the countenance, are general and characteristic appearances of a patient bearing a pancreatic cyst. The appearance of the abdomen depends entirely upon the size and direction of the growth of the tumor. A tumor must attain considerable dimensions before its presence can be detected by inspection. Larger tumors may produce an abdominal distention equal to a full-term pregnancy. Though usually spoken of as an epigastric tumor, the enlargement occupies chiefly the umbilical and left hypochondriac regions. I have found it extending upward into the epigastrium and across to the right side, or, in case of a large tumor, appearing as a symmetrical distention down to the pubis. The characteristic feature, however, is that the history, when obtainable, will show that the enlargement commenced above and somewhat to the left side, and grew from that site of origin. In emaciated subjects the surface of the enlargement may change in appearance according to the degree of distention of the stomach. Distinct colonic patterns may appear along the lower border of the tumor, creating a suspicion of an intestinal origin of the growth.

Palpation: Under palpation we note that the tumor is smooth, elastic, often fluctuant, generally movable with respiration, and in some instances, as recorded by Doran and Israel, extremely mobile, may be pushed well back into the loin of the left side. Such latitude of mobility is exceptional; usually the tumor is more or less fixed, permitting slight lateral displacement. It is often pulsatory. Pulsations of the splenic artery have been felt upon the anterior surface of the tumor; and in one instance the aorta could be both seen and felt in front of the tumor.

Percussion: It is by percussion chiefly that we define the location, boundaries, and relations of the tumor. As Abbe has

shown, a pancreatic cyst may develop, and rise above the stomach; it may lie entirely concealed under and behind the stomach; it may lie under or below the colon; or, as is usual, it lies between the stomach and the colon. Inflation of the stomach with carbonic-acid gas and percussion will accurately outline the relations to the stomach. Percussion without inflation may be deceptive, as the empty stomach may be compressed between the tumor and parietes, and yield a dull percussion-note. A tumor of sufficient dimensions will show dulness below the stomach and above the colon. The relation of the colon is developed by distending it with water; the percussion-note is not as flat over the bowel as over the surface of the tumor. Of 63 cases studied by Harris to determine the relation of the colon to pancreatic cysts, in 34 it was displaced downward; in 3 it was along the upper border; and in 7 the colon passed directly across the surface of the tumor. In 19 other cases no mention was made of the relation of the colon, yet from the operation done it must have been displaced downward. He thinks it safe to conclude that in 95 per cent. of cases the colon assumes a definite downward relation. In pseudocysts especially, Mr. Lloyd has called our attention to an area of dulness and impaired breath-sounds in the left back. In such cases there is usually upward displacement of the apex-beat of the heart. In moderate-sized cysts, percussion is the most valuable means of diagnosis, but in very large cysts that reach to the pubes, crowding the intestines laterally and downward into the pelvis, percussion is of but little value, and we must rely for diagnosis upon the history of the case, the site of development of the tumor, and upon the digestive disturbances.

Paracentesis: The examination of the contents of the cyst after aspiration is uniformly mentioned as a diagnostic test. Serious symptoms have followed the simple introduction of a hypodermic needle into a pancreatic cyst; death has resulted from aspiration. Keen and Doran especially condemn exploratory puncture. Selected seemingly-dull areas, when exposed by celiotomy, have proved to be occupied by empty flattened-out stomach or intestine. Large blood-vessels may be punctured. But aside from these dangers, the microscopical and chemical analyses of the fluid, while fairly conclusive, are not infallible. Von Jaksch has pointed out the existence of a ferment in ascitic

fluid and in the contents of other cysts of the abdomen, which has slight power of converting starches into sugar; and furthermore, the contents of pancreatic cysts may not have the power of emulsifying fats.

SUMMARY OF SYMPTOMS.—The history of an injury or of attacks of epigastric pain, digestive disturbances, emaciation, with sudden development of a tumor occupying the epigastric, left hypochondriac, or umbilical regions, approaching the anterior parietes, and lying between the stomach and colon; the rapid growth of the tumor, sometimes sudden increase in size; its extremely fluctuating character, and often its close relation to the back, as is shown by dulness over the lower costal region behind, are the characteristic symptoms upon which we make the diagnosis of pancreatic cyst.

DIFFERENTIAL DIAGNOSIS.—**Tumors of the kidney** grow from the loin forward and downward, usually maintain their kidney-shape, can generally be pushed upward and backward to disappear in the loin. The relation of the colon to its inner border in a small tumor is definite. Digestive disturbances are inconspicuous symptoms. There is usually marked alteration in the quantity and character of the urine.

Ovarian Cyst: Of 18 cases of pancreatic cysts collected by Fenger, 6 were diagnosed as ovarian cystomata. In their incipiency ovarian tumors are usually painless, and digestive disturbances occur only when pressure-symptoms become marked. Emaciation is not necessarily present, jaundice is a mere coincidence, diabetes may exist as a complication. More important, however, is the primary seat of development from the pelvis. Ovarian cysts with long pedicles may prove most confusing, but the absence of general symptoms, and the relations of the tumor should make the differentiation.

Echinococcus of the Liver: The comparative good health of the patient, the slow growth of the tumor, its continuity with the liver, and its movements with respiration are usually sufficient to define the character of the tumor.

Distended Gall-Bladder: The position of the tumor in the right hypochondriac or umbilical regions, its relation to the liver, its mobility in the arc of a circle, often the unimpaired health of the patient, and the history of biliary colic with its characteris-

tically referred pain, with or without jaundice, are the distinguishing features.

In **abdominal aneurism** the pulsation is expansile; in cysts it is not. If we place the patient with a pancreatic cyst in a knee-chest position, the transmitted aortic beat disappears.

Omental cyst moves freely with respiration, may be displaced upward and laterally, but not downward, occupies the region of the umbilicus or below; the abdomen is peculiarly pendulous; percussion shows the superficial location of the tumor, and areas of resonance are not encountered over it. The paroxysms of pain are of short duration, and are attributable to weight and traction. The stools do not indicate the disturbance of pancreatic function.

PROGNOSIS.—The course of pancreatic cysts is usually rapid. They may be slow in their early development, but when of sufficient size to be recognizable the accidents of hemorrhage and inflammation usually lead to rapid changes. Untreated, the prognosis is very grave. Death is attributable to functional disturbances, inanition, hemorrhage, or peritonitis.

SURGICAL INDICATIONS.—Efforts to remove the entire cyst are usually unsuccessful and fraught with danger. In view of the position impressively taken by Mr. Lloyd, that many of these cysts are effusions into the lesser peritoneal cavity, the operative treatment should consist in incision and drainage. Persistent fistulæ may prove annoying, but, as a rule, closure occurs after several weeks. McBurney prefers to do the operation in two stages, and this gives the best operative prognosis. Bessel-Hagen states that in 19 of the 149 cases total extirpation of the cyst was done, with 17 cures and 2 deaths.

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ABSCESS OF THE SPLEEN.

DEFINITION.—A suppurative inflammation of the spleen. Cases are recorded as splenic abscess in which the suppuration was perisplenic.

SYNONYM.—Suppurative splenitis.

FREQUENCY.—Primary suppuration of the spleen is a very rare affection; I am able to gather 3 cases in addition to the 7 collected by Bessel-Hagen.

ETIOLOGY.—Metastatic foci of suppuration in the spleen are not unusual, and, as such, they are expressive of a pyemic state and are not amenable to surgery. Primary suppuration occurs usually in an organ predisposed by engorgement, either from displacement with torsion of its pedicle, or in hypertrophy from paludial or other systemic infections. Traumatism does occasionally result in suppuration in a previously healthy spleen, but usually there is some predisposing cause, as is evidenced in Sendler's case of a girl four years of age, who for some weeks had been languid, had lost appetite, and had diarrhea; in this weakened condition she suffered a fall, the brunt of the force being sustained over the splenic region; some weeks later she developed a splenic abscess, which was relieved by operation. It may occur without any recognized etiology.

PATHOLOGY.—The organ is irregularly enlarged, discolored, its capsule thickened, and there is secondary perisplenitis, with adhesions to viscera and parietes. The entire pulp-substance may be consumed in the degeneration. The abscess, though in apposition with the parietes, shows but little tendency to rupture externally. A doubtful case, reported by Happel, was pointing in the left lumbar region. It may rupture into the peritoneal or pleural cavity, stomach, colon, or, as noted by Grand-Mourse1, into the splenic vein or vagina.

SYMPTOMS.—The clinical course of splenic abscess is obscure and difficult to follow. The history of the case, revealing an assignable cause, with or without predisposing conditions, will mate-

rially aid in the early recognition of suppuration in the spleen. The cases usually present a septic chart and appearance; there is rapid emaciation, diarrhea, digestive disturbances, and a condition of marasmus. A spontaneous pain and tenderness, with increased area of dulness in the splenic region, are the special points of diagnosis. In a few instances in which there are adhesions to the parietes there may be the local evidences of inflammation, an appreciable tumor, and overlying edema; fluctuation may be elicited in large accumulations of pus. While there are no examinations recorded upon which to base the statement, yet in splenic abscess, perhaps more than in suppuration in any other portion of the body, there will be marked leucocytosis; and unless the local signs of inflammation are present, the detection of leucocytosis with enlarged spleen introduces a new and confusing feature in the diagnosis.

Differential Diagnosis.—From *malarial hypertrophy* it may be distinguished by the acuteness of the course, the irregularity of the enlargement, the evidences of perisplenitis, the negative results of medicinal treatment, and finally by the absence of pigment and malarial parasites from the blood.

Renal and **perirenal suppurations** occupy a deeper situation, excite pain in the lumbar, sacral, and genital regions, are covered by intestine unless very large, and are separable from splenic dulness by a zone of resonance. The history of the case and urinary analysis will further relieve the difficulties in differentiation.

Prognosis.—Expectantly treated, abscess of the spleen has a fatal prognosis, barring the accidents of spontaneous evacuation. Surgically treated, of the 10 cases noted recovery followed in all.

Surgical Indications.—If the spleen is fixed to the parietes by adhesions, *splenotomy* is, perhaps, the safer operation. Lauenstein, after exposing the spleen by incision, opened the abscess with cautery, and stitched the capsule of the spleen to the abdominal wall; the patient recovered.

Unless adhesions are insurmountable, *splenectomy* is the preferable procedure. Of the 7 cases collected by Bessel-Hagen of splenectomy for abscess, all recovered.

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ECHINOCOCCUS OF THE SPLEEN.

DEFINITION.—A cystic disease of the spleen due to a larval form of the tenia echinococcus.

SYNONYM.—Hydatid cyst of the spleen.

FREQUENCY.—Coen finds 58 cases of solitary echinococcus cyst of the spleen recorded in literature, and adds 3 of his own.

ETIOLOGY.—An infection by the embryo liberated by the swallowed ovum of the tenia. It may reach the spleen through the lymph-channels or by penetration of tissues.

PATHOLOGY.—Any portion of the organ may be the seat of deposit. The cyst may attain an enormous growth, and shows a tendency to suppuration or calcification. It may rupture into the peritoneal or pleural cavities, or into adjacent viscera. Continued growth of the cyst disturbs the continuity of and destroys the splenic structure, elongating the attachments of the organ by its weight.

SYMPTOMS.—Sometimes there are no symptoms other than the tumor. In other cases the symptoms are grave, and are chiefly due to pressure, adhesions, and traction upon the splenic ligaments.

PHYSICAL SIGNS.—The tumor is irregular, sometimes multilocular in feel; in large cysts decided fluctuation is felt, and a hydatid thrill may be observed. The tumor prolapses from the normal position of the spleen, occupies the left and lower portion of the abdomen, and may reach into the pelvis. It is usually fixed by adhesions, and is not movable under manipulation or with respiration.

DIFFERENTIAL DIAGNOSIS.—The differential diagnosis should be made from the history, symptoms, and physical signs. Exploratory puncture should not be resorted to, as it is not free from danger. A hydatid cyst cannot be distinguished from a simple serous or blood-cyst. The parasitic nature may be suspected if there are great adhesions.

From *ovarian cystoma* the diagnosis should be made by the history of the enlargement, its mode of growth, and the finding of the uterine appendages not connected with the cyst.

If suppurating, it cannot be distinguished from abscess of the spleen, except by history of its long duration.

PROGNOSIS.—Huber estimates the duration of this process in the spleen at ten years, though many cases may pursue a much more rapid course. Recovery may follow from sacculation, suppuration, and discharge externally or into the intestines. Recovery is recorded from evacuation by route of the lungs (Coen).

SURGICAL INDICATIONS.—Echinococcus cysts of the spleen may be treated by *splenectomy,* when the enlargement is of moderate size and adhesions are surmountable. The danger of this effort is rupture of the sac and peritoneal contamination. Bessel-Hagen reports ten cases of splenectomy for hydatids within the last decade, with only one death. The usual method of treatment, and perhaps the safest procedure, is laparotomy, suture of the sac to the parietes, incision, and drainage.

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SIMPLE CYSTS OF THE SPLEEN.

Simple cysts of the spleen, from their origin in extravasation of blood within the capsule, are usually spoken of as blood-cysts. Schalita describes a tumor as large as a man's head, which, he believed, had its origin in an aneurism of a branch of the splenic artery. In Subbotié's case the cystoma, which attained to the size of a man's head, developed after a severe attack of intermittent fever. Both Péan and Wells operated, believing that they had to deal with an ovarian cystoma. The subjective symptoms are indefinite. The diagnosis must rest upon the seat of development, direction of evolution, and the continuity of the tumor with splenic dulness. A cyst developing in a previously displaced spleen would offer the greatest difficulties in diagnosis.

Bessel-Hagen collects 7 cases of small cysts of the spleen with sanguinolent contents, all recovering after *splenectomy*.

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MALARIAL HYPERTROPHY OF THE SPLEEN.

DEFINITION.—Enlargement and pigmentation of the spleen in chronic malaria.

SYNONYMS.—Malarial spleen, "ague-cake."

FREQUENCY.—Malaria is distributed through a wide geographical range; it prevails more frequently in tropical and the warmer temperate climates, and exists epidemically in swampy regions, along the banks and deltas of great rivers. Among the residents of such localities it is quite common to find "ague-cake" or enlargement of the spleen.

ETIOLOGY.—Malarial splenic hypertrophy occurs as a sequel of repeated acute malarial infections or in chronic malarial cachexia, and is attributed to the deleterious influence of the hemocytozoa of malaria upon the blood. The parasite of the estivo-autumnal fevers is most usually associated with this phenomenon of splenic enlargement.

PATHOLOGY.—When one considers the grave anemias which, of necessity, follow frequently repeated attacks of malaria, the result of the degenerative changes which occur in the blood-forming organs, it is not surprising that we should find its chief pathological expression in the spleen, which organ is usually found enlarged, may reach below the umbilicus, to the pubes, and even into the true pelvis. The enlarged spleen is firm and has distinct borders. There is frequently perisplenitis forming adventitious attachments. Its capsule is thickened, showing fibrous plaques. On section it is slate-colored, and trabeculæ are prominent.

Thayer, who quotes largely from Bignami, thus describes the formation of the splenic hypertrophy: There is an aggregation in the pulp of the spleen of great numbers of red blood-corpuscles, which are shrunken and decolorized; they are included in colorless elements of the spleen as brassy-colored fragments or hyaline masses. A large number of the red corpuscles contain parasites. There is an accumulation of colorless elements which contain pigmented red corpuscles that are often necrotic. As a

result of these processes, a considerable number of the proper elements of the spleen become necrotic, while others, as well in the pulp as in the follicles, undergo karyokinetic division; this is followed by hyperemia of the splenic pulp. "The splenic pulp is crowded with phagocytes, some of which are small and similar to mononuclear leucocytes, while others are extremely large. . . . These cells, which may attain an enormous size, are laden with pigment, either in the shape of large blocks and clumps, or in small spheres or rodlets or very fine granules." When the acute infection has passed and the hyperemia subsides, changes take place in the necrotic areas of the splenic pulp, the vessels become dilated, forming a net-work of venous lacunæ. There may be extensive areas of cavernous sinuses. The follicles undergo hyperplasia, attain sometimes to three or four times their normal size; there is hyperplasia of the pulp around the follicles, and great thickening of the reticulum. There is widespread deposit of melanin throughout the connective tissue and pulp, but not in the follicles. "When one considers," says Thayer, "that each new infection is accompanied by fresh processes similar to this, it is easy to appreciate the gradual development of enormous splenic tumors, in which sometimes it is difficult, even histologically, to recognize the original structure of the organ."

The blood will show the characteristic pigment, but not necessarily the malarial parasite, unless examined during a febrile attack. The crescentic and ovoid forms of the parasite will probably be found, if the infection is of the estivo-autumnal character. There is decrease of red blood-corpuscles, a decline in hemoglobin. The number of white corpuscles in malaria is almost invariably subnormal.

Symptoms.—Residence in malarial districts, or history of recurring malarial outbreaks is a significant anamnesis. Some patients present a conspicuous anemia amounting almost to a cachexia, with grayish-yellow color of the skin, tendency to edema of the extremities, muscular weakness, and dyspnea. Gastro-intestinal disturbances, especially a persistent and annoying diarrhea, are common features. Vespertine or irregular temperature is not infrequently encountered. Other individuals may have escaped, in a great measure, or have passed through the

FIG. 1.

FIG. 2.

USUAL POSITIONS OF COLON IN RENAL AND SPLENIC ENLARGEMENTS.

FIG. 1, Renal tumor showing usual displacement of colon.

FIG. 2, Splenic tumor overlying colon.

toxic stage, and appear in perfect health, presenting as their only symptoms the enlarged spleen and distress due to the mechanical effects of its increased size.

The tumor presented by a spleen enlarged from malaria may be only sufficient to show a moderate increase in the normal outlines of the organ, or it may produce a swelling which will occupy the entire abdomen, resembling a full-term pregnancy or an immense uterine fibroma. The enlargement is usually asymmetrical, lying chiefly to the left side. Not infrequently the organ is displaced so that both poles may be felt. Its edges are sharp, and its right border notched; left lateral and posterior surfaces convex, right lateral and anterior, flatter. If of moderate size and not attached by adhesions, it will appear to change its form; at times it is smooth and flat, and again it will rise up as a sharp ridge; these alterations in form are due to rotation upon the pedicle, and are usually accompanied by pain, nausea and vomiting; and in one instance that I have observed, there was great depression, amounting to shock, which was relieved when the organ spontaneously assumed its normal relations. If very much enlarged, it may be felt in the pelvis, displacing and compressing the pelvic organs. If of moderate dimensions and not wandering, it will move with respiration. Unless fixed by adhesions, a hypertrophied spleen of moderate size may be freely moved upward and backward, in the direction of its normal residence. Unless displaced, the upper end is concealed by the costal margins and cannot be reached. It rests in close apposition to the parietes, and is uniformly dull upon percussion. A venous hum is not infrequently heard over it. The superficial abdominal veins are often enlarged, and the outlines of the liver are usually increased.

DIFFERENTIAL DIAGNOSIS.—The diagnosis of malarial hypertrophy of the spleen rests upon the history of malarial toxemia and upon the blood-examination. The presence of pigment and the detection of the parasite should distinguish it from primary splenomegaly. The normal or subnormal number of leucocytes eliminates splenic leukemia. The size of the spleen and the comparative good health of the patient are sufficient usually, without the blood-examination, to discredit the existence of pernicious anemia.

PROGNOSIS.—The moderately-enlarged, soft spleen due to acute malarial infection will yield to medical treatment. The indurated, hypertrophied spleen of chronic malaria may exist for many years without causing serious symptoms. It is exposed to the accidents of injury, displacements, and torsion of the pedicle. Laveran and Jonnesco do not hold that the spleen is the tomb of red blood-corpuscles bearing parasites, but that it is a receptacle or habitat of the malarial organism. Those patients having enlarged spleens and resisting malarial treatment are cured of their chronic malaria and anemia by the removal of the organ. "To remove the spleen," says Jonnesco, "is to remove the breeding-place."

Immediately after removal of the spleen leucocytosis develops, which may be several months in disappearing. After splenectomy I have observed decided pyrexia, great weakness, rapid wasting, marked anemia, restlessness, delirium, vertigo, and copious intestinal hemorrhage. These symptoms continued for several days, gradually subsiding, and in two months the general condition of the patient was excellent, the blood almost normal.

CONTRAINDICATIONS TO OPERATION.—The size of the organ is not a contraindication to operation. Extensive adhesions, according to Bessel-Hagen, are the most positive contraindication. Vanverts found that in 39 cases of enlargement with marked adhesions 28 died, while in 35 without adhesions only 2 died.

SURGICAL INDICATIONS.—A moderately enlarged spleen which does not give rise to pain or other symptoms does not demand removal. If pressure- and traction-symptoms are marked, if anemia is progressive, if diarrhea is uncontrollable, if the great mobility of the spleen renders it liable to torsion, or if malarial symptoms are persistent, we should then resort to *splenectomy*. Bessel-Hagen collects 61 cases operated upon since 1891, with a mortality of 23.4 per cent. Jonnesco has personally operated 28 times for malarial hypertrophy, with 8 fatalities.

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PRIMITIVE SPLENOMEGALY.

DEFINITION.—A primary endothelial hyperplasia of the spleen with progressive anemia.

SYNONYMS.—Primary splenic anemia, splenic pseudoleukemia.

FREQUENCY.—The condition of hypertrophy of the spleen with progressive anemia without increase in white corpuscles, which are the essential features of the disease under consideration, has recently received much attention. The different pathological findings, owing chiefly to the stage of the disease in which the specimens were obtained, and the various theories of the etiology, have led each ambitious observer to designate it according to his own peculiar ideas; consequently the subject is befogged with a manifold nomenclature. Banti's disease, that is, splenic hypertrophy with hepatic cirrhosis, is clinically pictured in some of Osler's cases of splenic anemia. The disease described by Gaucher, and later by Bovaird, primary splenomegaly, presents the same features as the splenic pseudoleukemia of Sippy. The clinical and pathological studies are yet too few to justify us in expressing positive conviction as to the identity of the several conditions, and until then we cannot determine its frequency. I find only 32 cases upon which to base my studies.

ETIOLOGY.—**Age**: In 1889, von Jaksch described a form of splenic hypertrophy as 'anemia infantum pseudoleukemica,' which, be it understood, is an entirely distinct disease from that under consideration. Bovaird holds that primary splenomegaly is necessarily a disease of childhood, and uses that as an argument in distinguishing it from splenic anemia as described by Osler. Waiving the contention that they are not one and the same disease, we find two cases of primary splenomegaly exhaustively studied by Brill, and four by Churton, all occurring in adults. On the other hand, several cases of splenic anemia in children are recorded.

Sex: It occurs with equal frequency in the sexes.

The etiology of primary splenomegaly is unknown. Neither malaria, tuberculosis, nor syphilis are satisfactorily assigned causes. Previous state of health has no predisposing bearing; many of the patients are apparently in perfect health when the enlarged spleens are discovered. It is unnecessary, and perhaps futile, to attempt to define the relation between the anemia and the hypertrophy of the spleen. Our knowledge of the physiology of this organ is too limited and uncertain to warrant us in speaking definitely upon its functional perversions. We know that, in all cases of primary splenomegaly thus far reported, the splenic enlargement precedes any marked anemia; that there is progressive deterioration of the blood with hypertrophy of the spleen. It is probable that the changes in both may be due to some toxic general condition; yet in those cases of marked primary splenomegaly accompanied by anemia, we have complete recoveries following removal of the spleen, which would indicate that the pathological changes in that organ were capable of inducing the profound alterations in the blood and general nutrition.

Pathology.— **Spleen**: The spleen is greatly enlarged, having been found in one instance to weigh twelve and a half pounds in a child whose total weight was only seventy-five pounds. The surface is generally smooth and the outlines an exaggeration of the normal, though there may be areas of fibrous adhesions. A cut section will show slight thickening of the capsule, and triangular pyramidal areas extending from the surface into the center of the organ will be observed. These are infarctions; they may be either anemic (white) or hemorrhagic (red), and vary in their appearance according to the extent to which organization or reparative processes have progressed. The structure of the infarct is that usually found in infarction of this organ. The trabeculæ of the spleen are somewhat increased in thickness, and when the organ is cut it offers a fibrous resistance to the knife. The general color of the organ is dark-red. But little change will be observed in the Malpighian bodies; upon microscopical section they may appear nearly normal, or may show an increased amount of fibrous tissue. Throughout the bulk of the organ the reticulum will be denser, with, in many areas, a great increase in the normally-present fibrous tissue; this may be espe-

cially marked around the blood-vessels. Moderate-sized areas of fibrous tissue may be found displacing portions of the pulp. The pulp-spaces are often dilated and filled with blood, and the endothelial lining gives evidence of some swelling. Hyperplasia of these endothelial cells has been observed, increasing the thickness of the lining walls of the spaces to several layers (Bovaird). Many of these cells may have desquamated, and are then found free in the center of the pulp-spaces, making a picture suggestive in its arrangement of the desquamated alveolar cells in catarrhal pneumonia. These cells may vary considerably in size, some of them having been found which from their size suggested epithelial cells; they may be polynuclear, vacuolated, or may present mitotic figures. There is probably no doubt, however, that these cells are of mesoblastic origin, and that the process may be characterized as an endothelial hyperplasia.

Blood: The hemoglobin is reduced, as is also the number of the red cells; in a series of cases the red blood-corpuscles averaged about three million per c.mm. The size and shape of the corpuscles are normal, as a rule. Irregular cells (poikilocytes) are either absent or present in very small numbers. Nucleated red cells are rarely found. The percentage of leucocytes is normal or slightly increased, rarely over 10,000 per c.mm., while the proportion of the various types of white cells to each other is about as is usually found. Pathological varieties of white corpuscles are generally absent. The specific gravity of the blood is diminished, its color is pale, owing to the reduction of hemoglobin, it coagulates rapidly, and the blood-platelets seem to be increased.

Liver: The liver may be increased in size, the amount of the increase depending somewhat upon the length of progression of the disease, cases being recorded in which it extended to the right iliac crest. The fibrous tissue of the organ, as well as the capsule, shows considerable increase in thickness. There may be increase in the size of the lymphatic spaces, and they, as well as some of the capillaries, may be found to contain a dark iron pigment (hemosiderin). In some places large cells are found in the capillaries and lymphatics, which resemble the large cells found in the pulp-spaces of the spleen; these have been thought to be true metastases brought to the liver from the spleen

through the portal vein; but the evidence seems to show that these cells in the liver are not true metastases, but that their origin is from a hyperplasia of the endothelial lining of the lymph-spaces and from the connective tissue of the liver itself (Bovaird).

Lymph-Glands: The lymph-glands for the most part remain normal throughout the course of the disease. In protracted cases it is not unusual to find the retroperitoneal and bronchial glands somewhat enlarged, singly or in groups. Glandular hypertrophy is always moderate, if present at all.

Symptoms.—Primitive splenomegaly is a disease of unknown origin, insidious in its onset, chronic and progressive in its course, and usually fatal in termination when treated medicinally. Prodromal symptoms are inconstant and variable. Muscular weakness, headache, epistaxis, and digestive disorders are occasionally noted in the previous history of patients, before the recognition of any organic changes. Usually it is the alteration in figure, the perceptible abdominal enlargement that leads the patient to consult a physician.

Enlargement of the Spleen: The several disorders of the spleen, which we have embraced under one head, and for want of pathological differentiation we consider as one, present in common as idiopathic hypertrophy of the organ, and this enlargement, being uniform, symmetrical in character, constitutes a true hypertrophy, which is one of the essential features of primitive splenomegaly. It is the first symptom to attract the patient's attention, and owing to the position of the spleen, it attains some considerable degree of enlargement before it is recognized. We are, therefore, unable to fix the date of the commencement of the pathological process which results ultimately in such pronounced morphology. The growth is gradual; when, however, the organ attains to sufficient dimensions, its increased weight, aided by gravity, tends to produce displacement, alters the relations with the blood-vessels, favors congestion, and facilitates rapid growth; it is, therefore, remarked that the latter stages of this disease are characterized by the most rapid growth. Usually the organ does not extend more than a hand's breadth below the costal margins, but in some instances it attains the enormous growth met with in true leukemia. The enlarged

spleen emerges from beneath the left costal arch, clings closely to the abdominal wall, develops downward and to the right, and overrides all structures in the abdominal cavity. It may be moved laterally, but by preference in a diagonal direction upward and backward from right to left. Its lower margin may be distinctly outlined, its sharp lower pole easily grasped. The left lateral and posterior surfaces are convex; the right lateral and anterior surfaces flatter in comparison. The right border is characteristically notched, which anatomical feature is not effaced in instances of greatest hypertrophy. Unless fixed by adhesions it responds to the movements of the diaphragm. It is uniformly dull over its surface; the dulness may reach to the seventh interspace. It is liable to the accidents of displacement with torsion of its pedicle. By traction it not infrequently excites pain. Tenderness is rarely pronounced. Disturbances of the stomach, large intestine, pancreatic and biliary secretions, are mechanical phenomena attributable to pressure or traction by displacement of their attachments, direct or indirect, to the spleen.

Enlargement of Liver: In certain forms of this disease there may be enlargement of the liver. Osler remarked it in a few of his cases.

Anemia: Anemia is an integral part of the pathology of this affection. It is not symptomatically expressed during the early stage of the disease. Osler says that more than one-half the cases, when they come under observation, show only slight evidences of anemia. Sometimes early, and sometimes not for several years, but inevitably at some time in the course of the disease the symptoms of anemia appear. The debility, loss of flesh, pallor of mucous surfaces, transient edemas, and vertigo are the early evidences of blood-depravity. The red blood-corpuscles may show 4,000,000, or more, the hemoglobin 75 per cent. to 80 per cent., with no increase of white corpuscles in this stage of the disease; but with the advance of the splenic hypertrophy the anemia progresses to the stage of cachexia, and the blood-examination usually shows a grade of anemia of a chlorotic type. In the advanced stage of the disease the red blood-corpuscles may be reduced to 1,500,000; hemoglobin is reduced to a very slight amount; but there is no leucocytosis presenting

the characteristics of leukemia. The skin is often bronzed, evening elevation of temperature is not unusual, profuse sweating and sudamina have been especially remarked by Bovaird.

Circulatory Disturbances: Hemic murmurs are often to be heard in the latter stages, the carotids throb, and the abdominal veins are varicose. Sippy describes the pulse as full and frequent; it is more often feeble and frequent. Vertigo and cardiac dyspnea are symptoms of sympathetic disturbances, as they occur before there is marked anemia or great enlargement of the spleen.

Hemorrhages: All authors have observed the frequency of hemorrhages from the mucous membranes, the gums, the nose; and in the cachectic stage petechiæ may be distributed over the body. But Osler especially emphasizes the significance of hematemesis and melena as symptoms of splenic anemia; they occurred in more than one-half of his 15 cases, and in some instances prior to the physical and microscopical evidences of anemia, and oftentimes early in the course of the disease and at varying intervals.

Digestive Disturbances: Nausea, vomiting, epigastric or left hypochondriac pain are noted as early symptoms in many cases. In others, digestive disturbances do not appear until the spleen attains sufficient dimensions to make pressure, and anemia and cachexia are well advanced. Diarrhea of an intractable character is not an infrequent symptom. The loss of appetite, the attacks of vomiting, and intestinal disturbances hasten the decline.

Ascites: Transient edema may occur at any time in the course of the disease, but peritoneal effusion does not usually occur until the disease is well advanced. It may be due to hepatic cirrhosis; it is more probably due to pressure and peritoneal irritation, the condition of the blood favoring the transudation.

The clinical picture of primary splenomegaly is characterized by its insidious onset, great splenic hypertrophy, slight hepatic enlargement, anemia of a chlorotic type at first, deepening into cachexia, but never approaching a leukemic form; pigmentation of the skin, capillary hemorrhages, gastro-intestinal disturbances, a cachexia which resembles that of malignancy, which phenom-

ena may all appear in a few months or be protracted through a course of years.

DIFFERENTIAL DIAGNOSIS.—**Splenic-Myelogenous Leukemia:** The clinical phenomena presented in this condition so closely resemble splenic anemia that from symptoms and physical signs we cannot make a trustworthy differentiation. We are forced, in making this discrimination, to rely upon the blood-examination alone. In both conditions there is a diminution of red blood-corpuscles; in both there is marked decrease in the per cent. of hemoglobin; in both the color-index may be normal or lower; in both poikilocytosis is slight. In splenic anemia nucleated red blood-cells are rare; in leukemia they are more frequent than in any other form of anemia. Under some complications which may be intercurrent with splenic anemia, we may have an increase in the number of leucocytes—otherwise the white-cell count is normal or only slightly increased; it is in splenic leukemia that we find an enormous increase in leucocytes, far greater than is met with in any other anemia. Myelocytes are rarely found in splenic anemia; the number of myelocytes, sometimes amounting to 60 per cent. of the leucocytes, and which Cabot found in 18 cases to average 37.7 per cent., so far exceeds the highest count of them met with in any other condition that Cabot thinks that the blood of splenic-myelogenous leukemia is absolutely peculiar and characteristic, and cannot be confused with any other disease.

Pernicious Anemia: In pernicious anemia the red cells may be 1,000,000 or less per c.mm., and often show increase in diameter; in primitive splenomegaly they average 3,000,000, often higher, yet in extreme cases, in the stage of cachexia, as in Case XIII of Osler's series, they may be reduced to 1,540,000, but are of normal diameter. In pernicious anemia there is a marked decrease in leucocytes; in primitive splenomegaly they are normal in number or slightly above. The hemoglobin in pernicious anemia is variable, sometimes relatively increased, with high color-index; in primitive splenomegaly there is relatively low hemoglobin, the decline keeping pace with the progress of the disease. Nucleated red cells are commonly found, often in numbers in pernicious anemia, the megaloblasts outnumbering the normoblasts; nucleated red cells are exceptional in splenome-

galy. Poikilocytosis is much more frequent in pernicious anemia than in splenomegaly. In pernicious anemia Cabot lays stress upon the fact that the red-blood-cell count may approximate the normal several times in the course of the disease, each time followed by relapse and a rapid fall to extreme oligocythemia; in splenomegaly the progress of the anemia is uninterruptedly progressive. Valuable as the blood-examination is, we need not rely entirely upon it for differentiation. The dead pallor of pernicious anemia is characteristic; in splenomegaly the pale color of anemia is displaced by a bronze-hue, or, as Osler observed in 6 cases, by a well-marked melanoderma. In all forms of anemia it is not unusual to find the spleen somewhat enlarged, but it does not attain to the gigantic dimensions observed in primitive splenomegaly. Under proper hygiene and hematics improvement will be noted in pernicious anemia; but in primitive splenomegaly all medication is practically ineffectual.

Malaria: The enlarged spleen or "ague-cake" not infrequently met with in Southern climes presents many features in common with primitive splenomegaly. The irregular temperature and chills, the dirty slate-colored hue of the skin, renal complications, the efficiency of quinine and arsenic, and the detection of the plasmodium in the blood, together with the blood-pigment in the white corpuscles, are points, when taken collectively, which exclude primitive splenomegaly.

Gastric and **Duodenal Ulcer:** The gastro-intestinal symptoms with hematemesis and melena suggest ulcer of the stomach or duodenum. The blood-examination and the enlarged spleen should save us from such an error.

PROGNOSIS.—The course of this disease may be characterized as progressive. Its duration may be many years, and patients may die of intercurrent trouble. The average duration, according to Sippy, is about two years. Osler takes a somewhat conservative view, and does not advise immediate surgical intervention. The unfavorable prognosis under medicinal treatment, and the absolute cures and low mortality from splenectomy, commend this method of treatment.

SURGICAL INDICATIONS.—In appropriately selected and accurately diagnosed cases *splenectomy* should be done.

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WANDERING SPLEEN.

DEFINITION.—A displaced movable spleen.

SYNONYMS.—Splenoptosis, movable spleen, prolapse of the spleen, splenectopia, floating spleen.

FREQUENCY.—Wandering spleen is a frequent complication of all forms of splenic enlargement; it participates in general enteroptosis, and occasionally occurs as an independent phenomenon.

ETIOLOGY.—The organ is maintained in its position chiefly by the phrenicosplenic and gastrosplenic ligaments. From its normal position it may descend because of increased weight, or from relaxation of its ligaments, or partial rupture of its supports from trauma. It is an acquired and not a congenital condition. It is rarely met with in men, frequently observed in multiparous women.

PATHOLOGY.—In prolapse of the spleen the traction upon the phrenicosplenic ligament is greatest, and may cause rupture of the suspensory ligament, or may elongate it into a thin cord. The spleen is then supported chiefly by the gastrosplenic attachment, and tends to assume a horizontal position in the abdomen, the hilum then being directed upward. The weight of the organ exerts traction upon the stomach, duodenum, pancreas, and bile-duct. The tail of the pancreas has been found drawn into the pelvis by the spleen. It may cause uterine displacements by its presence in the pelvis or by increased intra-abdominal pressure.

With descent there is a tendency to rotation of the organ; the mechanism of the rotation is probably to be explained by peristaltic action of the intestines and by gravity in changing posture of the individual. It has been found rotated upon its pedicle by six distinct turns (Stone). The effects of rotation are chiefly due to strangulation of the circulation of the spleen and pancreas. Slight constriction affects the venous circulation chiefly, leading to enlargement with liability to rupture. Gangrene and peritonitis may result.

An inflamed wandering spleen may acquire attachments to the intestines, kinking and obstructing them; or from this source

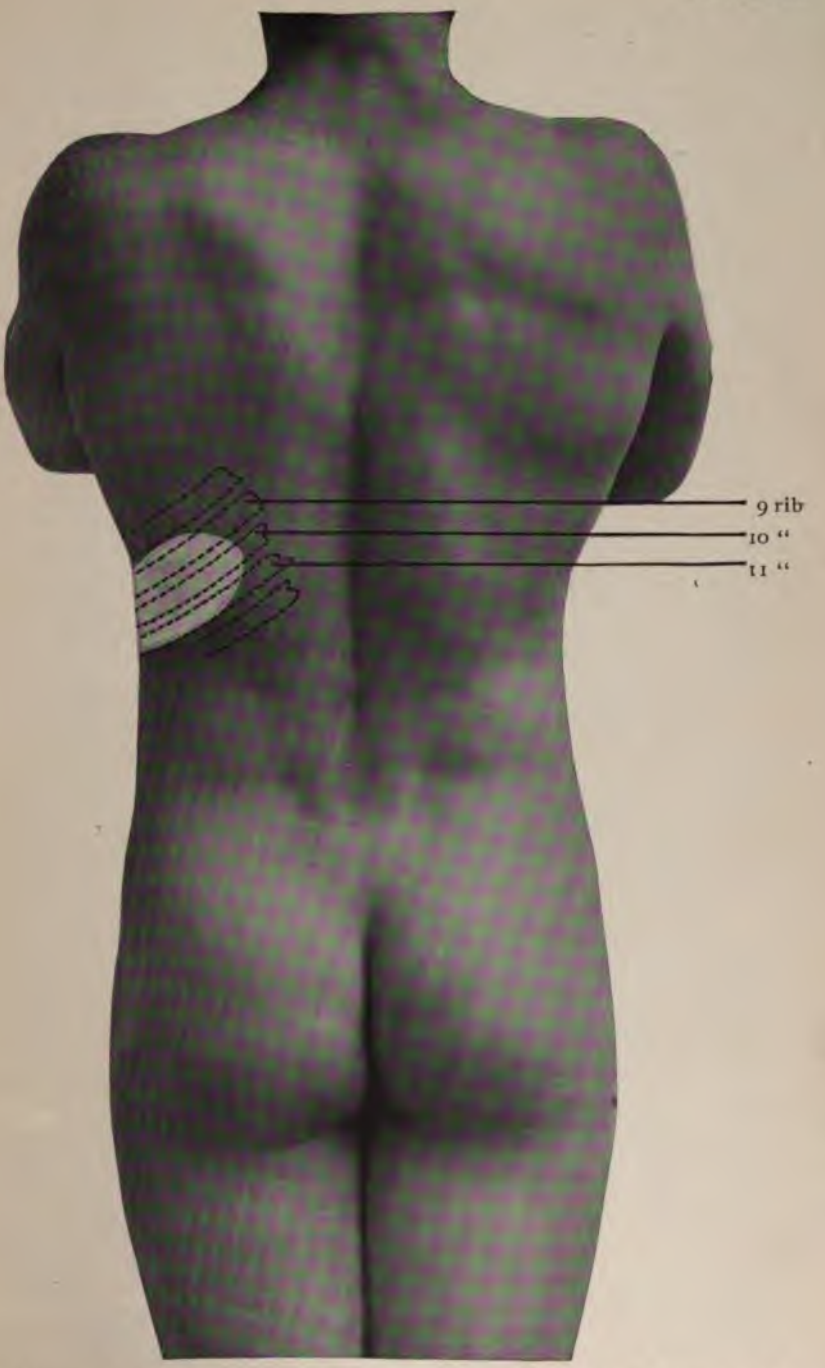
it may receive infection, splenic abscess resulting. The cases of atrophy of displaced spleens, which have been reported by Viard, may have been due to gradual torsion of their pedicles and obstruction of their blood-supply. The chief effects of wandering spleens are attributable to traction upon the stomach and pancreas. Displaced spleens are usually enlarged; the hypertrophy may be either a cause or an effect of the displacement; yet wandering spleens of normal size are found.

Symptoms.—The symptoms of wandering spleen, as one would naturally expect, proceed chiefly from traction and pressure. Gastric disturbances, nausea, vomiting, a dragging pain in the epigastric and left hypochondriac regions, a sense of weight and heaviness, are complained of. These patients are usually thin and gaunt, suffer from diarrhea, may occasionally be jaundiced from obstruction of the common duct through traction on the pancreas; and menstrual disorders have been observed in association with movable spleen. They are conspicuously nervous; conscious of a movable organ, they grow apprehensive and melancholic.

The symptoms of wandering spleen become interesting and urgent when the organ rotates upon its pedicle. When this happens the pain is severe, cramping, paroxysmal in character, and felt chiefly in the left hypochondrium. Vomiting is persistent and shock marked. The pulse becomes feeble and fast, respiration restrained and shallow, abdomen hyperesthetic and quickly tympanitic, bowels constipated, and urine scanty. Urso says that the most important symptoms of torsion of the pedicle are sudden, acute anemia following the rapid onset of symptoms, and the increase in the size of the spleen. In one case that I observed the spleen apparently doubled in size, and the patient died in collapse in six hours from the onset of symptoms. If the rotation be partial the organ may spontaneously right itself, and all symptoms will then subside. The symptoms after rotation depend upon the completeness of the strangulation.

The normal area of splenic dulness is oval in shape, its long axis corresponds to that of the ribs, and it extends from the eighth interspace to the eleventh rib, between the mid-axillary and mid-scapular lines. The detection of a tumor of splenic shape in the left half of the abdomen should lead us to determine

PLATE VIII.



Position of Spleen and Its Relation to Ninth, Tenth and Eleventh Ribs.

immediately if the spleen is in its normal position. While wandering spleens are usually enlarged, they maintain their general splenic contour. They may lie horizontally across or in the vertical axis of the abdomen, they may even be displaced to the opposite side, or may rest in the bottom of the pelvis. A greatly displaced spleen of moderate size will not respond to the movements of respiration; but where the spleen is only slightly displaced or very much enlarged, unless fixed by adhesions, it will obey the movements of the diaphragm. Uncomplicated, it is freely movable, chiefly in an upward and backward direction; and when it has escaped from beneath the ribs its borders may be outlined, it may be grasped between the hands, and its shape and dimensions well shown against the relaxed abdominal wall. Its sharp anterior festoon-border is characteristic and diagnostic. In rotation of the organ upon its pedicle this border may lie under and behind, and so be nonpalpable. In its descent it lies in apposition to the anterior parietes, overlies the colon and intestines, and is, therefore, dull over its anterior surface. Adherent coils of intestines may occasionally give areas of resonance. These physical signs, with the absence of the organ from its normal position, determine the splenic nature of the tumor.

DIFFERENTIAL DIAGNOSIS.—**Movable Kidney:** A wandering spleen may be mistaken for a movable kidney. The superficial position of the tumor, the shape of the organ, the absence of urinary symptoms, the possibility of palpating the kidney in its site, and the absence of the spleen from its normal position, suffice to distinguish the two conditions.

A displaced spleen was diagnosed by Polk as a pelvic abscess. Klein records a case in which a wandering spleen was found lying in the vesico-uterine pouch; it had been diagnosed as an anteversion of the uterus, and a pessary was found in the vagina. It has been repeatedly mistaken for a pedunculated uterine fibroma. These errors are scarcely pardonable. Careful investigation as to the seat of origin of the tumor, a study of its physical characteristics, and a thorough examination should disclose the true nature of the tumor.

Greater difficulties of diagnosis confront us when there is torsion of the pedicle. There may be no history or knowledge of enlargement of the spleen, the symptoms develop suddenly and

present a striking similarity to intestinal obstruction. The detection of the characteristic spleen-shaped swelling, its rapid increase in size, the conspicuous, acute anemia, and the possibility of obtaining bowel-action by means of enemata should make the differentiation.

PROGNOSIS.—Wandering spleens are not necessarily productive of symptoms. When enlarged they usually give rise to distress. The danger lies, first, in the condition causing the enlargement; second, in the accidents of torsion and inflammation, in rupture of the pedicle, stomach or duodenum, or of the spleen itself, and in intestinal obstruction.

SURGICAL INDICATIONS.—A wandering spleen giving rise to distressing pain, or affecting the general health by reason of visceral disturbance, should be removed. Rapid increase in the size of the organ or great mobility should indicate the danger of complications and should urge to surgical interference.

The operative mortality of *splenectomy* for wandering spleen in carefully selected cases is very low. In 43 cases operated upon in the last decade there were only 3 deaths (Bessel-Hagen), results which commend the radical operation over uncertain splenopexy. Schwartz removed a hypertrophied and wandering spleen from a woman, 36 years of age, in the fifth month of pregnancy; gestation was not interrupted.

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SARCOMA OF THE SPLEEN.

Primary malignant neoplasms of the spleen are exceedingly rare. They occur most frequently as lympho- or round-celled sarcomata. It is questionable if primary carcinoma of the spleen has ever been met with.

The tumor usually results in a symmetrical enlargement of the spleen, resembling the simple hypertrophies or enlargement from leukemia. The growth is rapid, metastases occur early, directly by the blood from the spleen; adhesions and secondary growths are, therefore, common complications. The diagnosis of primary malignant disease of the spleen is exceedingly difficult during life, and it is not improbable that malignant spleens have been removed and the specimens mistaken for splenomegaly. There are no characteristic symptoms or signs that enable us to determine the malignant character of the splenic tumor. The disease appears to be more common in adults and in women. Billroth has the distinction of removing the first malignant neoplasm of the spleen, in 1884, recorded by Von Hocker. In malignant disease of the spleen the blood shows marked leucocytosis, characterized by an increase in the eosinophile cells and by an absence of polymorphism. It is chiefly upon the blood-examination and the rapid splenic enlargement that we are able to exclude leukemia.

Bessel-Hagen collected 5 cases of sarcoma of the spleen removed by operation prior to 1890, with a mortality of 40 per cent.; since then 4 cases, with a mortality of 25 per cent. Two patients lived after removal of the spleen 2 and 4 years respectively. Extensive adhesions greatly increase the dangers and difficulties of the operation, and darken the postoperative prognosis.

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TUBERCULOSIS OF THE SPLEEN.

Tuberculosis of the spleen, as a primary infection, is admittedly rare. Hamilton records a case observed in the Presbyterian Hospital, Chicago. Of twelve cases of tuberculosis in children, in ten the spleen was the site of chief manifestation (Manicatide). In all the spleen was enlarged. The tubercles involve chiefly the Malpighian corpuscles and pulp, and are sparsely distributed in the interstitial tissue. There is thickening of the capsule, with perisplenitis. The organ is enlarged and tender, usually displaced. Emaciation, pyrexia and diarrhea are active symptoms. The progress of the disease is usually rapid.

Splenectomy is indicated only in the early stage and in the absence of other localization.

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ANGIOMA CAVERNOSUM OF THE SPLEEN.

I find two reports of this unusual growth of the spleen. In Hunter McGuire's case, recorded by Hoge, in a woman, æt. 45 years, loss of flesh, alternating attacks of diarrhea and constipation, with the development of a tumor in the left side, which reached to the pelvis, were the symptoms which called for operation. Recovery was prompt, and when last heard from the patient was in excellent health.

Homan's patient was a female, æt. 22 years; when first seen she was very anemic, with a pulse of 142. The abdomen had been enlarged for over a year, and had been tapped upon five different occasions; at each tapping seven to ten quarts of bloody fluid were removed. At the first operation, a year after the first tapping, seventeen pounds of bloody ascitic fluid were removed from the abdomen, and an omental tumor and a supernumerary spleen were ablated. Four months later, a second operation was done; several flattened masses were found in the mesentery, the spleen was found enlarged and was removed. The patient survived this operation only a short time.

In cavernous angioma the spleen is dark, soft, and highly vascular. Microscopical examination shows that the capillary system is lost, and that the blood is contained in a number of freely intercommunicating spaces, which resemble the corpora cavernosa of the penis. The walls are formed of a thin connective tissue with an epithelial lining, and where the wall is formed by splenic tissue proper there seems to be a slight thickening, as if there had been a preëxisting space.

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RENAL CALCULUS.

DEFINITION.—A free concrement formed in the interstitial, parenchymatous, or pelvic portion of the kidney.

SYNONYM.—Nephrolithiasis.

FREQUENCY.—Renal calculi may be voided spontaneously, may occupy secondary positions, or may remain symptomatically quiescent. Therefore, any statistics as to their supposed frequency are valueless.

ETIOLOGY.—**Age:** It is generally stated and accepted that renal calculus occurs most commonly at two periods of life, in children under fifteen years of age, and in individuals over fifty years. In this country it is most frequently met with between the ages of thirty and fifty years and is quite unusual in children.

Sex: Of 90 patients subjected to surgical procedure, 32 were females and 58 were males, which figures perhaps indicate the relative frequency in the two sexes. Morris, combining his statistics with those of Tuffier, finds that in a total of 406 cases there were 218 males and 185 females. There is no satisfactorily assigned reason why it should appear in the one sex more than in the other. The ratio 3 males to 1 female, usually given in the text-books, shows a predilection for males which does not exist.

The conditions, predisposing and exciting, which give rise to the formation of renal calculus are by no means clearly defined. Climate, character of food, water, occupation, have each in their turn and all together been held as influences sufficient in themselves to lead to the formation of renal concretions. All modern writers reject as inconsequential such predisposing causes. Upon the presumption that calculi were due merely to a precipitation of inorganic elements, which exist normally in the urine, but for some reason were in excess, the formation of such calculi was attributed to the constitutional condition supposed to be responsible for this excess of salts. Such a state was designated a 'diathesis.' Many of the highest authorities maintain

that heredity exerts a strong influence in the production of calculus. Against these theories of heredity and the constitutional origin of renal calculus Newman long ago brought forth incontrovertible argument. Uric-acid stones do not necessarily form in conditions in which those crystals are in excess; for instance, in leukemia, in which there is an enormous excess of uric acid in the urine, calculi are rarely formed; and on the other hand, such calculi may form when a diminished amount of uric acid is being eliminated (Harris).

The examination of uric-acid infarctions in new-born infants and of the calculi themselves furnishes information as to their etiology. After dissolving the inorganic elements, Ebstein found that there was an organic substance, albuminous in nature, in which the solid particles had been entangled. Scharling and Ord believe that this colloid cement-substance is necessary for the formation of calculi. This substance or stroma is the product of the mucous membrane largely, and consists of mucus, débris, and epithelium, the result of inflammation, the "stone-formation" catarrh of von Hemsbach. This is the organic base essential to the formation of all free concretions.

The detection of bacteria in the center of renal calculi, first made by Galippe in 1886, is an observation now common in concrement-formation; and the inference is natural that the micro-organisms excite the catarrhal inflammation, the products of which favor crystallization and precipitation, the minute aggregation growing by accretion. It is very clear that, following infection, bacterial decomposition of urine does take place; and in such instances it is proper to ascribe to the microörganisms the initial and essential part in the formation of calculus. Baginsky asserts that in infants suffering from intestinal trouble he has found masses of colon-bacilli in the pelvis and canals of the kidney, and in consequence of their action has seen a pyelonephritis. In Lennander's case, in addition to the stone, was found a brownish mass composed of a fibrinous, albuminous body, urates, coloring matter of the urobilin group, and zoögleæ of colon-bacilli. It is an unproved assumption to accredit the formation of stones to bacteria without infection of the kidney. That these microörganisms can filter through the uriniferous tubules, calices and pelvis, exciting no pathological change in

the kidney, and yet give rise to stone-formation, is attributing to them a pathological result that they nowhere else in such manner initiate.

The presence of a foreign body in the kidney or its pelvis does not necessarily result in the formation of a concrement. Maas's observation upon the effect of traumatism with hemorrhagic effusion into the renal substance would indicate that around a blood-clot there would be no crystalline deposit unless there was inflammation. Tuffier's experiments upon animals showed that there was no stone-formation as long as asepsis was maintained. These facts are certainly conclusive that inflammation and decomposition of urine with formation of ammonio-magnesium phosphate give the conditions necessary for the formation of phosphatic calculi, but do not explain the pathogeny of the other varieties.

Renal calculi are composed of substances that are normal constituents of the urine, and these substances need not be present in abnormal amount; hence stone-formation cannot be said to be due to a diathesis. It would appear that the only essential is the presence of a colloid cement-substance, without which any solid ingredients of the urine would not cohere. The presence of the albuminoid matter is abnormal, and must be due to inflammation, which in turn may be due to any one of several causes, perhaps most frequently to bacterial infection.

Pathology.—Renal calculi may be homogeneous in structure, or distinctly stratified in arrangement. They are named from the inorganic elements which enter into their composition, as uric-acid stones, phosphatic, oxalate-of-lime, cystin, etc. They are classified as simple and compound; simple when containing only one constituent of the urine, compound when composed of two or more. The classification of Albarran and Dickinson into primary and secondary stones, in view of recent etiological studies, is no longer desirable. The kind of calculus usually varies with the age of the patient; Mr. Taylor found the urate of ammonium so frequently the nucleus of calculus in infancy that he named such concretions the 'infantile calculus'; in adult life the uric-acid concrement is the most usual variety, while in advanced age the formation is of the oxalate of lime.

The **uric-acid calculus** is the most common of all varieties. It varies in size from that of a grain of sand to that of a goose-egg. Its weight varies from 10 to 1,000 grains, though it is reported that Reynier removed by operation a calculus of this variety which weighed 9,750 grains. The surface is smooth or granular, the color dark-yellow or red. It may be simple in structure, or show a uric-acid nucleus encased in a shell of earthy phosphates.

The **oxalate-of-lime stone** is usually dark-brown or greenish in color, almost black, generally of rough surface, and very hard, often showing a nucleus of uric acid.

The **phosphatic calculus** is irregular in shape, whitish or grayish in color, usually soft and friable.

The **cystin stone** is soft, waxy, and brownish-yellow.

The **xanthin** variety is red, smooth, and friable.

Compound stones, as a rule, have an outer rind of the triple phosphates.

Location of Stones: Calculi may occupy any part of the kidney. Uric-acid and urate concretions form chiefly in the tubular structure of the kidney, while the other varieties usually occupy the calices. The shape of the stone depends upon its seat of formation, upon its composition, and upon the number of stones. When several stones lie together they are faceted. When they occupy the renal pelvis they are branched and irregular, and often form a fairly accurate cast of this cavity and the calices. Such calculi, being usually of phosphatic composition, are friable, and may spontaneously undergo fragmentation. A movable stone is generally smooth and round. The stone-formation occurs in both kidneys in 20 per cent. of cases.

Number of Stones: In about 50 per cent. of the cases there is only a single stone.

The changes that occur in the kidney incident to the presence of a calculus are due to the pressure of the foreign body, inflammation, and obstruction to the renal urine. These changes do not follow as an immediate sequence of the calculus-formation. A stone of large size, irregular and mammillated, may remain imbedded in a calyx or in the substance of the kidney many years, giving rise only to limited and insignificant changes. If the calculus-formation is large or multiple, the atrophy resulting

PLATE IX.

NEPHROLITHIASAS; KIDNEY AND PART OF URETER.

A. Spiculated oxalate calculus in upper part of greatly dilated pelvis and calices. The stone consists of four bosses each almost covered by miliary bosses or spicules. Three of the bosses are shown in the drawing; one above and one below the leader from A, and one directly to the right of the end of the leader. The remaining projection is on the other side of the stone and extends upward into a dilated calyx.

B. A smaller conical calculus; the base only of the stone is shown, the apex extends into a calyx.

C. Greatly dilated calyx resulting from pressure, obstruction and irritation by the lowest boss of the calculus A.

D. Ureter which, at the end, has been dissected free from the peri-ureteral, newly-formed fibrous tissue that surrounds it and extends up over the dilated pelvis. The moderate but long continued inflammation of the mucosa of the pelvis and ureter has given rise first to an interstitial ureteritis and pyelitis which later has extended to the juxtaposed connective tissue inducing what might be termed chronic productive peripyelitis and peri-ureteritis which in turn have led to more or less firm attachment to adjacent structures.

from pressure may destroy the entire renal parenchyma. Every stone, however small, is surrounded by a limited zone of atrophy and induration, which may become the seat of fatty or malignant changes. Small calculi, movable in the pelvis, excite irritation, resulting in nephritis or pyelitis, abrading the mucous membrane, and causing minute hemorrhages. Such a stone may occlude the ureteral orifice and cause a retention of urine in the kidney. This condition of hydronephrosis is only exceptionally due to stone, yet it may occur, and the kidney be converted into a large multilocular cyst, the calices forming the septa. The secreting substance of the kidney rapidly atrophies and is absorbed after complete ureteral obstruction, and the resulting cyst is then not so large as when the stone intermittently obstructs. Usually the calculus excites such irritation as to invite mixed infection; if in the substance of the kidney, a renal abscess follows, which may open into the pelvis and imperfectly discharge through the ureter, or it may perforate the capsule and appear as a perirenal suppuration, and the stone may wander through this opening to be discharged through a mural fistula. A perinephric abscess may become subdiaphragmatic, but usually the pus burrows into the loin or inguinal region, and may then be mistaken for appendicular suppuration, but the true pathogeny of many perirenal suppurations is appendicitis. A stone in a calyx or the pelvis of the kidney abrades the mucous membrane, by pressure-necrosis causes ulceration, forms an atrium for infection, and the pyogenic organisms supplied by the blood or urine give rise to suppurative pyelitis, suppurative pyelonephritis, and pyonephrosis. The pelvis of the kidney may contain the stone and be primarily infected, but the tendency of this infection is rapidly to extend and to destroy the renal substance, a disastrous consequence attributable not only to the infection, but to the partial or complete occlusion of the ureter by the calculus or tissue-débris, and by inflammation of that duct itself. The retained and decomposing urine and pus accumulate in the kidney, a condition designated pyonephrosis. If the process is acute, which it only exceptionally is, the rapid distention may lead to rupture of the capsule or pelvis of the kidney into the bowel, peritoneal cavity, or surrounding retroperitoneal tissues. Usually a pyone-

phrosis from calculus is chronic, the fibrous and fatty capsules of the kidney become thick and acquire adhesions.

Stones lodged in the lumen of the ureter are passed from the calices or pelvis of the kidney; they never form in the ureter. Sharp small stones are more likely to lodge than perfectly round smooth ones. The obstruction may be partial or complete. Schenck, in tabulating 84 operative cases of ureteral calculi, found the position of the stone cited in 81 cases. In 19 the calculus was arrested within 2⅔ inches of the kidney, in 8 at or near the pelvic brim, while in 41 (over 50 per cent.) it was within 2 inches of the vesical mouth of the ureter. If the obstruction is partial, intermittent hydro- or pyonephrosis with degeneration of the kidney will follow; if absolute, rapid atrophy takes place, should the patient escape anuria. The effect upon the ureter is to produce rapid dilatation; it may attain to the size of the colon. The walls become thickened by inflammation and adhesion. Strictures may be seen in the canal, indicating stations of temporary lodgment of the stone. By ulceration a stone may migrate from the ureter and be discharged either into a viscus, or, an abscess having formed, escape externally through a fistula.

Symptoms.—A renal concrement may form, exist, and pass from the body symptomlessly; or it may reside quiescently for an indefinite period in the kidney. We must not forget the post mortem finding quoted by Doran, in which there were 24 kidneys containing stones, 11 only having had symptoms, 13 without. It is of far greater importance that we should appreciate that this secretive behavior often conceals a progressive pathological process. In a general way, it may be said that a stone grows in importance as it approaches the pelvis (Morris); that movable stones give rise to a greater variety of and to more severe symptoms than fixed or imbedded calculi; and that smooth stones excite less pain than rough ones. All this is, in a measure, true, but the exceptions are so frequent as almost to constitute the rule. Nephrolithiasis is essentially a chronic condition; its manifestations may be insignificant, obscure and misleading, generally intermittent in character, sometimes distressingly acute, and when complicated by infection or obstruction, imperious in nature. The symptoms are so variable and so often referred to other organs that the first difficulty we encounter is in determin-

ing whether or not it is the kidney that is affected. Having reached this conclusion, we are further embarrassed by the knowledge that the symptoms of other renal conditions closely simulate those of renal calculus. Conclusive as the Röntgen ray may appear to be in the detection of calculus, the author would advise against the acceptance as conclusive of any one symptom, sign, or crucial test in any condition; they are often fallacious, and if harmful in no other way it discourages analytical study of the special symptoms, every one of which must be considered in the diagnosis of renal calculus, and to the particularization of which we now proceed.

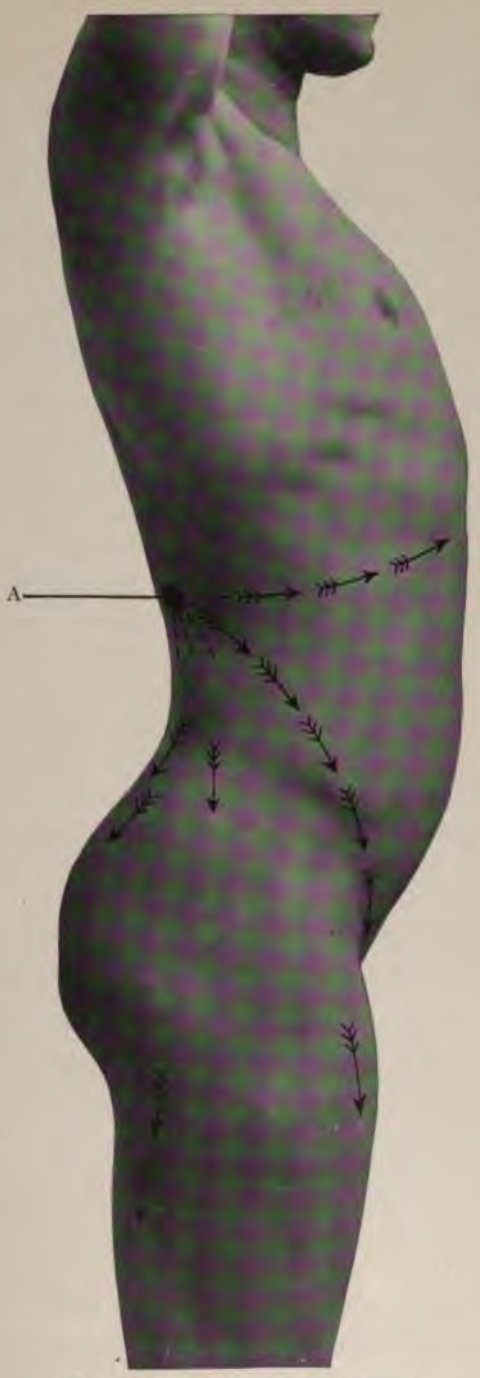
Pain: Pain, the most common symptom of nephrolithiasis, is a product of several different factors: the irritation of the calculus itself as a foreign body, the ureteral obstruction, and the accompanying inflammation may separately or in combination be responsible for the pain. A stone firmly fixed in the cortical substance of the kidney may not produce pain; but if loosely encapsulated, especially if it is irregular and spinous, pain may be the only symptom of such a calculus. A smooth stone, movable in the pelvis or a calyx, will excite great pain, whereas a rough one, fixed in any position, does not necessarily do so. The intensity and quality of the pain depend more on the location and mobility of the stone than upon its shape. A calculus, large or small, which obstructs the ureteral orifice, leads to a renal urinary retention and to sudden distention of the kidney. In such instances the pain is that of distention, a continuous, aching pain, accompanied by great tenderness. If the calculus becomes engaged in the ureter the character of the pain is now paroxysmal, and the suffering perhaps more intense and more unbearable than that met with in any other human ailment. When a calculus, resident in the pelvis or substance of the kidney, is attended by inflammation, the pain is always continuous, due to the accumulating products of inflammation, which distend the pelvis or the capsule of the kidney, obstruct the ureter, and prevent the outflow of urine.

Renal pain is either direct or referred. When confined to the immediate region of the kidney, it is said to be direct, and as such it may be sharp and lancinating, a dull ache, or it may appear as a stiffness and lameness; sometimes produces scolio-

sis; or else it is frequently described as a lumbago, and is then felt or complained of only when the body is in a strained or stooping posture. The pain is usually intermittent, *excited by exertion or exercise, and relieved by rest in a recumbent position.* These direct renal pains may exist as a symptom of renal calculus intermittently for years, and assume importance only upon the occurrence of obstruction or inflammation. The pain of renal calculus is not always confined to the kidney affected, and indeed may be felt exclusively in parts remote from the kidney. In renal calculus pain is reflected downward, usually in the course of the ilio-inguinal nerve to the genitals, and inner side of the thigh by the genitocrural nerve. It is also felt in the abdomen, in the sacrum, and in the course of the sciatic nerve to the sole of the foot. Exceptionally only is renal pain reflected upward. Vesical irritation may be sympathetic or due to a descending inflammation. Renovesical reflex is a common phenomenon; frequent and painful urination may lead us into ridiculous error. The pain in the genitals may sometimes be inflammatory in character, especially in children, when orchitis without assignable cause occurs as a symptom of renal calculus. Abrahams reports instances of inflammation of the testicles and ovaries occurring in nephrolithiasis, causing confusing symptoms. It is asserted that in calculus-disease of one kidney the pain may be referred and felt solely in the sound kidney, thus leading to operative error. This was a warm question some years ago. Mr. Morris strongly and clearly discountenances this teaching, holding that the seat of pain indicates the seat of disease. Other means of diagnosis now employed enable us to determine the involved kidney by more reliable symptoms than that of pain.

The above character of pain, direct or referred, may exist intermittently or continuously throughout a period of years preliminary to acute developments, and may constitute prodromal or suggestive symptoms of calculus, when suddenly the stone, dislodged from a cavity in the kidney, or moving in the pelvis and becoming engaged in the ureter, excites renal colic. In other cases renal colic may be the very first symptom of calculus. Lennander objects to the term 'renal colic,' and says that it should be called 'ureteral colic'; Rovsing, also, assumes that

PLATE X.



RENAL PAIN AND ITS DISTRIBUTION.

A. Area of irritation.

The arrows indicate some of the points to which the pain may be referred.

it is due to painful contraction of the ureter, and that it is therefore misnamed. Muscular spasm of the ureter is, no doubt, the chief element in this form of colic, excited by the foreign body; but that intense, paroxysmal pains do occur without engagement of the stone in the ureter, or the passage of any foreign material, is the author's observation. Hence some other factor, either sudden congestion or inflammation, must be considered. An attack of renal colic is not pathognostic of calculus-disease. The passage of a blood-clot or the débris of suppuration will excite it; "showers" of uric acid, the phosphates, or the oxalates may cause the paroxysms of pain; movable kidney is liable to accidents which may simulate the passage of a stone. Yet the pain of renal colic from calculus is usually unmistakable.

Renal colic may appear suddenly, either in the day or night, following exertion or in repose. The pain is first felt in the lumbar region of the side affected, passes downward along the course of the ureter to the bladder and genitals. The suffering is horribly severe; though paroxysmal in character, intermissions are not complete. The patient is thrown into a condition of shock, occasionally is nauseated and vomits, becomes pale, perspires freely, pulse is soft and feeble; in his agony he may faint or pass into convulsions; strong men have been known to attempt suicide. Pressure relieves the pain somewhat, hence the patient assumes a flexed position. There is a great desire to micturate, and a few drops of highly-colored urine, perhaps mixed with blood, are passed. In less severe cases there may be a copious discharge of urine from stimulation of the other kidney. The testicle of the affected side is tender and retracted. Tenderness develops over the kidney and along the course of the ureter. The paroxysms may suddenly subside with the passage of the calculus into the bladder, when there will be a free discharge of the urine, and the patient will express immediate and positive relief; he may be conscious of something having passed into the bladder. If the calculus is engaged in the renal orifice of the ureter, it may become dislodged; the relief attending this retreat of the calculus is great, though not so decided as is experienced when it makes a successful transit of the ureter.

When colic, the pain being of greatest intensity in the lumbar and lateral regions of the abdomen, is attended by the phenomena of vesical disorder, genital pain and oliguria, with perhaps hematuria, its renal nature is undoubted; yet there are cases in which the vesical, genital, and urinary phenomena are absent or inconspicuous, but in which the gastro-intestinal symptoms assume such prominence that Sternberg suggests that we call it the gastro-intestinal form of renal colic. Vomiting, tympany, constipation, and muscular rigidity are the misleading phenomena. These cases closely simulate appendicitis, and no less an eminent surgeon than William White has made the error. Gastro-intestinal symptoms are regarded by Dr. Leonard as significant of the ureteral position of the stone.

An attack of renal colic may terminate after a few minutes of suffering; it is usually measured by hours; sometimes exacerbations and remissions of pain recur for days. A reoccurrence of pain after a calculus has passed may be due to other calculi in transit, or if moderate to the irritation in the ureter. With complete obstruction of the ureter and arrest of the stone, the intensity of the pain subsides. Complete anuria may follow from sympathetic disturbance of the other kidney, or else the healthy kidney will assume the duties of the afflicted one, and the calculous kidney will take on the changes of hydro- or pyonephrosis.

Hematuria: A calculus imbedded in the substance of the kidney is encased in a fibrous zone, and does not give rise to renal hemorrhage. The bleeding occurs from the pelvis or a calyx of the kidney, and is due to the injury to the mucous membrane wrought by the stone, and so blood-casts are not to be found in the urine. It therefore follows that hematuria is more common from rough, irregular stones, especially the oxalate variety. The blood is not so intimately mixed with the urine as in structural disease of the kidney, and will separate and precipitate from the supernatant urine.

Hematuria may be an early symptom of calculus, but for reasons above stated it is usually a late manifestation. It is an intermittent phenomenon; it appears generally after violent exercise, may follow a rude physical examination, and occasionally appears during or after an attack of renal colic, though there is no necessary relation between the two. Copious and frequently

recurring hemorrhage would indicate a small stone, rough and movable, situated in the renal pelvis or passing through the ureter. The hemorrhage is rarely copious; it usually requires microscopical examination for detection. When blood is discovered in the urine, the physical appearance of the urine does not indicate its origin; its renal nature is only definitely determined by cystoscopic examination, when, if copious, the bloody urine may be seen escaping from the ureter. The quantity of blood is, as a rule, so small that this method of examination is not sufficient. The separate urines from each ureter must be obtained by segregation or ureteral catheterization, and microscopically examined. Such an examination, if affirmative, determines the question as to which kidney is involved. The presence of blood in the urine is significant but not conclusive of calculus-disease.

Pyuria: Pus in the urine is an evidence of infection. Its source in calculus-disease is usually the pelvis of the kidney. It is more constantly associated with renal colic, and is, perhaps, more frequently in evidence than is blood. When once it appears in the urine, unlike blood, it is likely to be found in all subsequent specimens examined, though it varies in amount. In the examination of the urine for pus the same precautions in technic must be observed as in the detection of blood-corpuscles. If urine which has habitually contained pus and blood should become clear of both, the presumption is that the ureter of the affected side is occluded, and only urine from the healthy kidney is flowing. A cystoscopic examination should be made to ascertain if this be true, though blocking of the ureter of a septic kidney will usually give rise to local and constitutional symptoms sufficiently grave to be diagnostic.

The above statements of the constant presence of pus and blood are not intended to be dogmatically made; clinically it is no unusual thing to find that both blood and pus may disappear from the urine without occlusion of the ureter, to reoccur with other symptoms of calculus.

Mucinuria: Reginald Harrison regards the appearance of a large quantity of mucus in the urine, in the absence of blood and pus, as a symptom of especial value in the early stage of renal calculus in children.

Urinary Reaction: Renal calculi are usually formed of uric acid or its salts, and of the oxalate of lime; and such formations are generally attended by acid urine. We have elsewhere referred to Albarran's classification of renal calculi into primary and secondary stones; the first class comprises those in which infection of the kidney has not occurred, and secondary stones are those which are formed in consequence of bacterial infection of the kidney. In the primary formation the urine is usually acid. Thomas B. Brown has studied 7 cases of renal calculus, 6 of which had alkaline urine, and 1 acid. The urine was obtained from the kidney by ureteral catheterization, and in the specimens that were alkaline the bacillus proteus vulgaris was found in 3 cases, the white staphylococcus in 2, and the sixth specimen was sterile. The chemical composition of the stone in all these cases was the same, the calculi being made up of the phosphates and carbonates of calcium and magnesium. He concludes that the microörganisms form the nucleus of the stone, decompose the urea of the urine, and produce the alkalinity necessary for this variety of stone. The specimen of calculus examined which showed acid urine was composed of uric acid and urates, the infection being due to the bacillus coli communis. These observations not only show the relationship existing between the variety of microörganism causing the infection, but it adds other cases to the list of those in which the calculus-formation is ascribed to bacteria, and it further shows that the chemical reaction of the urine depends upon the character of infection.

Thamuria: Vesical irritation, as is evidenced by frequent micturition, is a sympathetic symptom of some value in the diagnosis of renal calculus. It occurs usually during the day while the patient is active, and subsides at night. The quantity of urine voided in each act is generally small. This point is of special value in differentiation from tuberculous kidney. The dysuria, a conspicuous and distressing symptom during a seizure of nephritic colic, is reflex in character.

Psychical Disturbances: Nervous and mental phenomena are not infrequent in the clinical history of renal calculus. They usually partake of the character of melancholia, hallucinations, coma or stupor. They cannot always be ascribed to uremic

poisoning, nor are they accompanied by the temperature of sepsis, yet they are probably toxic in nature.

Palpation: Calculus-disease does not ordinarily produce enlargement of the kidney unless complicated. In hydro- and pyonephrosis, the result of ureteral obstruction, the tumor may develop after a severe paroxysm of pain, and gradually disappear; this may repeat itself several times throughout the clinical history, a symptom of great diagnostic value, and only simulated by movable kidney. Occasionally the stone may attain to such dimensions as to render it easily palpable; or if the concrements exist in multiple formation, friction-fremitus may be elicited. In palpating a kidney the seat of calculus, if the stone is producing irritation in the cortex, or if pyelitic infection is present, tenderness may be marked both in front and behind. Deep percussion will sometimes develop a sharp, stabbing pain, a sign esteemed of much value by Lloyd, and upon which as a guiding symptom Brook made the diagnosis of renal calculus in two cases.

Ureteral Sounding: The detection of a renal calculus lodged in the pelvis or ureter by observing the scratch-marks made upon wax-tipped ureteral bougies or catheters is a feat accomplished eleven times by Howard Kelly; by his admission it is affirmative evidence only. There are no symptoms by which we can distinguish renal calculus that is obstructing the renal orifice of the ureter from a calculus lodged in the ureter. The diagnosis of ureteral calculus rests upon this test of sounding the ureter, and the Röntgen-ray may reveal it. Exceptionally a ureteral calculus can be detected by vaginal or rectal palpation, or be felt through the abdominal parietes. In several instances the stone has been seen through the cystoscope protruding from the vesical orifice of the ureter. It is usually discovered after operation and failure to find the stone in the kidney, the catheter or sound then being passed from the renal orifice downward. If the due preliminary examinations of the urine are made before operation, a ureteral calculus will be detected. The frequency of their lodgment in the ureter has been only recently disclosed by X-ray examination.

Röntgen-Ray Examination: It is claimed that clinical experience has demonstrated that by the Röntgen ray in expert hands an absolute diagnosis, both positive and negative, can be made.

We are especially indebted to Dr. Charles L. Leonard, of Philadelphia, for the development of this mode of physical examination. In 1907 he reported 356 cases with only three per cent. of error. In his last one hundred cases there was only one error. The value of the X-ray as a means of early diagnosis of calculus varies with the skill of the skiagrapher and his ability to read his plates. In doubtful cases stone may be discovered and its destructive effects averted. No skiagraphic examination is complete unless both kidneys and both ureters throughout their length have been pictured. The Röntgen method will enable the surgeon to early operation; furthermore, it renders simple and precise the technic, since we have the stone definitely located from the skiagraph, and the incision can be made directly down upon it. It should always be employed in suspected cases when available, but excellent work has been and will be done without it; and it should not overshadow the clinical diagnosis. In justice to clinicians, I must record a personal experience with the X-ray. My patient, a robust man whose normal weight was 215 lbs., gave indefinite symptoms of renal calculus. He suffered with insomnia, tachycardia, phosphaturia, and lost 90 lbs. in two months. A skiagraph was made by no less an expert than Dr. Charles L. Leonard. It revealed a shadow which Dr. Leonard considered either a stone or an incrustation in the pelvis of the kidney. Immediate operation was done, the kidney opened from pole to pole, and thorough exploration of the kidney and ureter was made with negative findings. Yet I am happy to state that there was immediate improvement in all symptoms. The patient's weight at the time of operation was about 120 lbs. Within six months he had recovered his former weight and normal condition.

Exploratory Operation: There are some cases in which all available evidence is inconclusive, and yet the symptoms warrant exploratory incision and direct examination of the kidney. After exposure of the kidney the stone may be felt by palpation, but this method must not be relied upon. Morris refers to a case in which nephrectomy was done, and after the kidney was removed and upon the table nothing could be felt, yet a calculus as large as a marble was imbedded in its substance. Needling, because of its simplicity and supposed freedom from danger, has been relied upon to detect the stone; it is uncertain, not free from

harm, and should not be employed. The kidney should be incised upon its convex surface and carefully examined with sound and finger for the detection of the concealed calculus.

DIFFERENTIAL DIAGNOSIS.—**Renal Tuberculosis:** The symptoms presented by renal calculus are closely simulated by those of renal tuberculosis. In renal tuberculosis an inherited predisposition, a history of genital infection, or suspected latent or healed tuberculous disease in other organs, with loss of flesh and anemia, are suggestive points. Polyuria is the symptom of greatest value in tuberculous kidney. There is not only frequency of micturition, both day and night, but there is an increase in the quantity discharged; while in renal calculus there is frequent micturition during the day, relieved at night, and the quantity of urine voided is not increased. Hematuria is copious in tuberculosis, the urine often containing blood-casts, is spontaneous and not induced by exercise; it is a constant symptom in the latter stage of the disease, and may appear as a very early symptom. The hectic condition of the patient, tendency to pyrexia and sweating, are more important symptoms in tuberculosis. In the latter stages of the disease when pyuria is present a cystoscopic picture is in itself almost diagnostic, denoting the descending infection. And, finally, we may rely upon the bacteriologic examination of the separate urines, and upon the inoculation-test of the urinary sediment on guinea-pigs.

Appendicitis: The gastro-intestinal form of renal colic has been mistaken for appendicitis. The absence of temperature, the presence of blood and pus in the urine, the alterations in the quantity of urine, the absence of a palpable tumor, the location of the pain and its reflections, should eliminate appendicitis.

Gall-Stone Colic: Pain in the epigastrium or right hypochondrium, referred to the back and reflected upward, is in itself so different from the downward-reflected pain of renal calculus that upon this alone we may, in a measure, rely. Gall-stone colic is attended by local tenderness upon point-pressure over the gall-bladder. In cystic-duct obstruction a tumor may develop, having the physical characteristics of a distended gall-bladder. In common-duct obstruction jaundice with clay-colored stools denotes the channel involved.

Renal Tumor: A tumor, the result of calculus-disease, except in acute obstructive cases, has a history, which may date back many years, of renal pain, alterations in the urine, and slight hematuria. Renal tumors are of rapid formation, their clinical history is brief, the attacks of pain occur usually with hemorrhage, and the bleeding is generally profuse. A calculous tumor is very tender; a neoplasm is not. A septic condition is frequently present in calculus; slight pyrexia may attend a sarcomatous growth.

Renal calculus must be differentiated from affections of the urinary bladder, prostate, and urethra. Digital, instrumental, and cystoscopic examinations are our means of positively drawing the distinction.

PROGNOSIS.—When we realize that in a considerable number of cases renal calculus is a bilateral disease, that it leads to atrophy, abscess, hydro- and pyonephrosis, that unexpectedly a fatal anuria may develop, we recognize that, apart from the terrible suffering that it entails, the patient is confronted by great danger. That a stone may remain as an inoffensive tenant of the kidney, and that it may pass *per vias naturales* is consoling but dangerous knowledge. The latency of the symptoms does not necessarily imply that the kidney-substance is escaping atrophy. The final awakening may reveal hopelessly irremediable conditions. Those cases which pursue their course without characteristic attacks of renal colic are especially dangerous because of mistakes in diagnosis or tendency to defer operative interference.

SURGICAL INDICATIONS.—The aim of the surgeon should be directed to the recognition of renal calculus in its early stage, when his patient is in good condition, and before there is destructive atrophy, infection, hydro- or pyonephrosis, perinephric abscess, fistula, or the deadly complication of calculous anuria. Persistent pain, either direct or referred, with renal tenderness, may be the only symptom, and is sufficient to warrant exploration. When pain, continuous, intermittent, or characteristically colicky, is coupled with vesical irritation and hematuria, the indications are clearer. To wait for the symptoms of obstruction, with intermittent pyo- or hydronephrosis, conditions which cannot occur without definite pathological changes in and serious impairment of the function of the organ, is to incur the necessity of unsat-

isfactory and incomplete surgery, or of very radical measures under adverse conditions.

As a preliminary to any operative procedure upon the kidney or ureter we should ascertain, if the means are at our disposal, whether the patient has two kidneys, and if so, what is the functionating capacity of the two combined and of each separately?

The first point can be determined by cystoscopic examination of the urinary bladder which will show whether or not two ureteral openings are present. The administration of indigocarmine will frequently aid in the discovery of the ureteral orifices through discoloration of the urine. The blue urine escaping from the openings leads to more ready recognition.

The combined functional capacity of the kidneys is determined by getting the cryoscopic index of the blood, by determining the amount of urea excreted daily and by the phloridzin test, which is now used almost exclusively instead of the methyline blue test. The individual capacity of the kidneys is determined by finding the cryoscopic index and urea content of the urines as collected by ureteral catheterization, and by the phloridzin test made with the ureteral catheters in place.

Cryoscopy: Cryoscopy, as expounded by Von Koranyi, is simply a method of determining the functional activity of the kidneys by finding the molecular concentration of the blood and of the urine as shown by their freezing points. Deficiency of renal function will lower the freezing point of the blood by increasing its molecular concentration, while it will raise the freezing point of the urine by diminishing its molecular concentration. The freezing point of normal blood is .56 degrees C. below that of distilled water, while the freezing point of normal urine with moderate ingestion of fluids is 1.2 degrees, or a little more than this, below that of distilled water. Should the freezing point of a patient's blood prove to be as much as .6 of a degree below, and the freezing point of his urine prove to be less than one degree below that of distilled water, a deficiency of kidney function is supposed to be established. A method of cryoscopic examination perfected by Kümmell and considered absolutely reliable by him, after having it tested in more than 1,200 cases, is as follows:

"The determination of the freezing point is carried out with Beckmann's apparatus. This consists principally of a very fine

thermometer with one hundred divisions, each of which is again divided into one hundred parts. The thermometer is immersed in a glass cylinder, in which the fluid to be examined is kept in constant motion by means of a platinum rod. The glass cylinder, with the thermometer and fluid, is placed in a freezing mixture at a temperature of minus four degrees Celsius. While being constantly stirred the fluid is undercooled. A moment then supervenes when the fluid congeals. By the change from a liquid to a solid state heat is liberated, which permits the column of mercury to snap up to a certain point, where it remains stationary for a time; this is the physical freezing point. After standing for a time the column of mercury again falls, and gradually reaches the temperature of the surrounding freezing mixture. If, now, one determines in a similar manner the freezing point of distilled water—the scale of Beckmann's thermometer is not numbered, and the zero-point is not fixed—and subtracts the figure obtained from the freezing of the solution (*i. e.*, the blood) from that obtained by the freezing of the water, the remainder is the figure which shows how much lower the solution freezes than distilled water. In the case of blood this difference is 0.56 degrees C. One says, in short, the freezing point is 0.56 degrees, and a special sign has been chosen for this, viz., δ, while Δ designates the freezing point of urine.

"Our method of examination of the blood is as follows: Under aseptic precautions a hollow needle is thrust into an artificially congested vein of the arm, and from 15 to 20 c.c. of blood removed. This is caught up in the glass cylinder used for the examination, and is defibrinated by stirring with the platinum rod, immediately after which the freezing-point is determined. In a second cylinder the freezing-point of distilled water is determined each time. This latter procedure we consider necessary, since the position of the mercury in the U-shaped Beckmann thermometer is subject to variations, and because the newer thermometer with a fixed zero-point does not appear to us to be reliable. With sufficient practice the time necessary for the examination is from fifteen to twenty minutes."

Kümmell considers the cryoscopic index of the blood of more value than that of the urine, and claims that it is not affected by heart disease, pneumonia, typhoid fever, abdominal tumors and other conditions that are considered by some to be sources of error.

He finds the freezing point elevated by fever and by anemia without indicating a disturbance of kidney function.

The total ureal excretion, which should not be less than 300 grains daily with the patient in bed and on low diet, is determined by the method of Esbach, which is too familiar to require description here.

The Phloridzin Test: Within from ten to twenty minutes after the subcutaneous injection of phloridzin sugar should be found in the urine coming from a normal kidney. Renal disease not only affects the amount of sugar secreted following the phloridzin injection, but also the time required to secrete it. Should as much as forty minutes elapse before the appearance of sugar in the urine coming from a kidney, that kidney is certainly unable to take upon itself the entire work of urinary excretion. The test for the individual urines is made as follows: The ureters are catheterized and the first urines obtained examined; then 1 c.c. of warm sterile one per cent. solution of phloridzin is injected subcutaneously. Beginning ten minutes later, the urine is examined every five minutes as it comes from the ureteral catheters for sugar. This test, combined with cryoscopy, gives satisfactory results in determining the functional capacity of the kidneys. Both tests should be used whenever it is possible to do so.

After thorough investigation of the urinary function we are prepared to approach the question of operation.

The special procedure indicated in the treatment of nephrolithiasis will depend upon the condition of the patient, the location of the stone, the pathological condition present, and the state of the other kidney. The kidney is exposed, we presume by the usual lumbar incision. The exposure should be sufficiently free to permit palpation of the organ *in situ;* and while palpation should not be relied upon for the detection of stones, if so found, it specifically directs our incision into the kidney.

From the convex border a free incision is made into the kidney, the pelvis and calices are explored with fingers or steel sound, and if stone is detected it is removed. After ascertaining the patulous condition of the ureter, if there is no infection, the kidney-wound may be approximated with deeply-placed cat-gut sutures. This operation of nephrolithotomy is restricted to the removal of renal calculi from kidneys free from manifest infec-

tion, and when the ureters have been tested and are known to be free from obstruction.

Rovsing and many others counsel against incising the pelvis of the kidney. The author has followed this advice, and has encountered the greatest difficulties in removing a stone from the infundibulum. Under similar conditions I would another time employ *pyelolithotomy*.

In suppurative conditions of the kidney complicating renal calculus the ideal nephrolithotomy may not be done. The patient may be in such a low state that we are only warranted in incising and draining the kidney, removing the stone if accessible. This simple operation of *nephrotomy,* used in the sense employed by Mr. Morris and Greig Smith, has a mortality of fully 50 per cent., very much higher than *nephrolithotomy* upon apparently healthy kidneys, which, according to Thorndike, has a mortality of 14 per cent. Morris has had only one death in 34 operations. We regard it as a safe and successful procedure.

In calculous pyonephrosis the question is presented, Shall we do *nephrotomy* or *nephrectomy?* It is a rule established by Mr. Morris that it is safer to drain calculous pyonephrosis than to execute an immediate nephrectomy, but in suppurative conditions with ureteral obstruction that cannot be relieved, the other kidney being known to be active and competent, the indicated procedure is nephroureterectomy. The now-known frequency of ureteral calculi, and consequent hydro- and pyonephrosis, makes the operation of exploration of the ureter an essential part of the technic of nephrotomy. We may not be warranted at that sitting in attacking a ureteral obstruction, yet we have gained information upon which we may predict the future behavior of the case.

Secondary nephrectomy is demanded for recurring attacks of pain, for persistent fistula, and for conditions indicative of chronic sepsis.

In the treatment of ureteral calculus, unless aided by the skiagraph in localizing the stone, the greatest difficulties will be encountered. If the stone is lodged in the vesical end of the ureter or near thereto, it may be removed with forceps through the cystoscope in the female, or through a cystotomy wound. Or if palpated through the vagina, it may be cut directly upon through

the vaginal vault and removed. Morris says that when the stone is impacted in the ureter near the lower end, but too far up to be reached by bladder or vagina, the sacral route should be employed; he has availed himself of this procedure upon two occasions with success. When the calculus is situated so that it cannot be reached in such ways, if the stone is located by the X-ray or by Kelly's wax-tipped catheter, the ureter should be exposed by extraperitoneal incision, opened by a longitudinal slit, the stone extracted and the duct closed with interrupted silk sutures, an operation known as *ureterolithotomy,* as especially advocated by Dr. Hugh Young, who collects 7 cases of this extraperitoneal *ureterolithotomy,* with 5 recoveries and 2 deaths. If the kidney is hopelessly involved in infection or atrophied from ureteral obstruction, the operation of choice is *nephro-ureterectomy.*

In all operations for renal calculus, intravenous saline solution should be administered at the conclusion of the operation as a preventive measure against postoperative nonobstructive anuria.

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CALCULOUS ANURIA.

Mr. Henry Morris's Hunterian lecture upon calculous anuria is a remarkable and classical production; to his teaching I am largely indebted for the facts herein set forth, and I am pleased to make due acknowledgement. The recent articles of Duplay and Reclus, and Cabot, all impress us that suppression of urine is one of the gravest complications of renal calculus-disease. In a patient who presents a history of renal colic, should suppression of urine occur, the anuria should be regarded as directly due to the stone. In exceptional cases there is bilateral occlusion of the ureters by calculi, yet calculous anuria occurs when the renal orifice of one ureter or its channel is occluded by a calculus, the other kidney being absent, atrophied or diseased. It is rare indeed for a healthy kidney to suspend secretion for any length of time simply through reflex irritation from stone in the opposite ureter; this fact should be kept conspicuously before us in the management of anuria.

It is more common in males than in females, and while more frequently met with after middle life, it occurs at all ages. The history of patients who develop anuria usually points clearly to nephrolithiasis. When it occurs suddenly and in the absence of a history of previous attacks of renal colic, it is probable that there is some congenital abnormality present.

The suppression of urine may be complete or incomplete. Legueu, from an analysis of 30 cases in which there were post mortem examinations, found that on the side last obstructed there was obliteration of the ureter in 23 instances, and calculi in the pelvis of the kidney in the other 7. On the other side there was congenital absence of the kidney in 3 cases, atrophy or other change caused by calculus in 20, and obliteration of the ureter in 6; in one case there was slight epithelial proliferation of the tubules.

Anuria may follow immediately upon an attack of colic or be preceded only by lumbar and iliac pain. Mr. Morris says that

in some cases, which present great difficulty in diagnosis, there is no preliminary pain. The symptoms of onset are usually distressing; there is great desire to micturate, with passage of only a small quantity, a few drops or few teaspoonsful of thick, bloody urine; the amount voided depends upon the completeness of the obstruction.

Calculous anuria may be divided into two stages: *first, the period of tolerance,* which lasts from 5 to 14 days, during which there may be no urine voided and yet the patient feels and appears quite well. Mr. Morris regards this fact as differentially diagnostic from suppression following shock, injury, or such as appears in Bright's disease or cholera:—in all the latter instances the symptoms of uremia develop quickly, and if the suppression is relieved the return of the normal secretion is gradual; in calculous suppression there are for a time, no uremic symptoms, and if the obstruction is spontaneously relieved, there is at once an abundant discharge of urine. If the condition of hydronephrosis precedes the development of anuria, the tolerant stage is more prolonged than in an undilated kidney. The anuria may not be absolute and continuous; there may be intervals in which a small quantity of urine is voided, but not in sufficient amount to relieve the system, though it may delay the symptoms of the *second or uremic stage.* And these symptoms as given by Morris are, first, a slow and full pulse which later becomes irregular; irritable vomiting, constipation, and meteorism; diarrhea, observed in other forms of suppression, is only exceptionally present in calculous anuria; the nervous symptoms, contraction of the pupils, muscular tremors, delirium and hallucinations, indicate fatal poisoning; the respiration becomes slow and irregular, the temperature falls and the patient dies in coma or convulsions.

Of 48 cases collected by Mr. Morris, not operated upon, 38 died and 10 recovered; of 49 subjected to operation, 24 died and 25 recovered, figures sufficiently conclusive. When confronted with the condition of suppression of urine, every available means should be employed to determine the presence of a ureteral or pelvic calculus, regardless of the previous history of the patient. The case should be treated by operation early in the tolerant stage. Cabot has succeeded in relieving his patient by celiotomy and massage of the kidney, yet we would hesitate to commend

so doubtful a procedure under such imperative conditions. The indication is to remove the stone by *nephrotomy* or *ureterotomy* if possible, and to determine positively that the ureteral channel is clear; and if this cannot be done, to provide loin-drainage.

At times it is difficult or even impossible to determine whether one or both kidneys require exploration, and if only one, which is the organ at fault? Watson advises bilateral nephrotomy at one sitting in such cases. This plan, however, is only justifiable when the kidney first explored is found with an unblocked ureter, or in such a damaged state that its secretion would be of no value to the patient. Otherwise, the patient should be put to bed after one kidney has been incised, and at least twenty-four hours allowed for the reëstablishment of the renal function before the other organ is invaded.

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WANDERING KIDNEY.

DEFINITION.—A kidney of loose anchorage; when displaced by deep inspiration, both extremities are palpable.

SYNONYMS.—Movable kidney, nephroptosis, ren mobilis, loose kidney. Floating kidney, often used synonymously with the above, should be restricted to those cases in which the kidney is provided with a mesonephron.

FREQUENCY.—All statistics bearing upon the frequency of movable kidney are based upon palpable kidneys of whatsoever degree. A kidney that is accessible to palpation has not necessarily trespassed beyond its physiological limits. Glénard's classification recognizes four degrees of mobility of the kidney: the first is that in which the lower pole can be felt during deep inspiration, but recedes with expiration; second degree, in which a greater portion of the kidney, but not the upper pole, can be palpated; third degree, the upper pole can be outlined on deep inspiration; and fourth degree, the entire organ is palpable during both inspiration and expiration. In the definition we have limited wandering kidney to that state in which the entire organ is palpable upon deep inspiration; yet it is admitted that lesser degrees of ptosis may occasionally give rise to symptoms. The kidney enjoys a normal mobility of an inch to an inch and a half, and we do not think that the finding of the lower half of the kidney palpable should imply a pathological state. If investigators had tabulated statistics of movable kidneys productive of symptoms, a more correct idea of the pathological frequency of the condition could be derived.

Küster states that 4.41 per cent. of patients have pathologically movable kidneys, a percentage very much less than the usual estimate, as we shall show, but certainly in keeping with our own observation. Harris, who may be taken as an extremist, says that 56 per cent. of women have distinctly movable kidney upon one or both sides. Edebohls, whose work is exclusively gynecological, finds that 20 per cent. of his patients have mova-

ble kidney; and Godart-Danheiux from observations upon 603 women found that 35 per cent. of them had movable kidney. It is not recorded what number of them had symptoms.

Etiology.— **Age:** Wandering kidney is incident between the ages of twenty-five and forty-five years. Comby records 18 cases in children, the youngest one month old. It must be borne in mind that in childhood the kidneys are normally more palpable.

Sex: Though not exclusive to women, wandering kidney is preponderatingly more frequent in them. The author has never met with pathologically movable kidney in the male except in association with splanchnoptosis. Statistics show that it is from 7 to 16 times more common in females.

Misplacement of the kidney and fixation of the organ in an abnormal position is usually a congenital condition. In 1,000 post mortems there were 24 cases in which Newman described the position of the kidney as distinctly abnormal. Roberts shows that fixed misplacement is more likely to occur of the left kidney than of the right. The kidney occupies some position near the brim of the pelvis. Many interesting cases are reported of pelvic malpositions. Mundé, and Tait have mistaken it for diseased tube and ovary. Newman, in his dissertation on renal abnormalities, refers to many instances of pelvic kidneys. It is not infrequently associated with other abnormalities. In these cases of simple misplacement of the kidney, the renal vessels are abnormally long or else of ectopic origin.

Floating kidney is a congenital condition; the mesonephron, gathered at the hilum, forms a pedicle around the vessels and ureter, and the kidney enveloped in peritoneum moves freely in the abdominal cavity. This condition is, perhaps, more common than Morris asserts; Clarke met with 2 cases out of 30 nephrorrhaphies. It must be taken into consideration in the surgical treatment, as an intraperitoneal operation is demanded.

Returning to a consideration of the causes of wandering kidney, it is well to allude briefly to the natural supports of the organ. First, and of greatest importance, is that indefinable but well-recognized factor of intra-abdominal pressure; second, the position of the kidney, snugly ensconced in the paravertebral spaces; third, the perirenal fascia, as described by Glantenay and Gosset; fourth, the reflection of the peritoneum to the liver and

duodenum or to the spleen; fifth, the blood-vessels; and last, and of doubtful importance, the fatty capsule. Sustained in its position by these several factors, it naturally follows that displacement and loosening of the organ will occur under two conditions: first, from loss of intra-abdominal pressure; and second, from weakening and elongation of its local supports. It so happens oftentimes that more than one element is active in the production of a displacement. Conditions under which there is loss of intra-abdominal pressure are usually attended by local changes, thus doubly favoring displacement.

It is generally stated that multiparous women with pendulous abdomens furnish the greatest number of subjects with wandering kidney. They present typical cases of lowered intra-abdominal supportive power. It is further argued by Newman and others that women of the poorer class, who are forced to resume their labors before the abdominal muscles recover their tone after confinement, are the cases in which wandering kidney is most frequently found. Admitting the force and correctness of this position, yet the frequency with which wandering kidney occurs in nulliparous women (40 per cent., according to Harris) lessens the importance of pregnancy as an etiological factor. My observations certainly accord with Deaver's, that wandering kidney is most common in neurasthenic nulliparous women. Greig Smith asserts that it is among the upper classes that he has met with it most commonly. Those who contend that the relaxed abdominal wall is responsible for the descent of the kidney instance its occurrence after the removal of tumors or the withdrawal of fluid.

We should not ascribe the ptosis alone to the loss of intra-abdominal pressure, but consider the peritoneal tension in these conditions. The frequency of wandering kidney in women is not chargeable alone to the relaxation following parturition, but is attributable to the displacements consequent upon injuries to the pelvic floor in parturition. Increased weight of the kidney, whether from tumor, calculus, or enlargement, leads to yielding of the supports and descent of the organ. French authorities, Lancereaux and Becquet, give menstruation as a cause of this affection, in that it produces or is attended by congestion of the right kidney; the periodically recurring increased weight of the

organ results in gradual relaxation of its capsule. To this cause we attach but small importance.

Chronic constipation is about six times as common in women as in men. Newman has pointed out that the ascending colon joins the transverse by an obtuse angle, or even with a dependent loop; fecal matter is pressed through this portion of the canal by peristaltic power only, and in chronic constipation there is constant traction in this locality. This tugging upon the peritoneum which is reflected over the upper pole of the right kidney is an element to be considered in causing its displacement, and, in some measure, it accounts for its frequency in females; and it further explains the frequency of displacement of the right kidney. Furthermore, the increased effort required at defecation heightens the intrathoracic pressure, which exerts itself upon the kidney.

Glénard maintains that movable kidney is always associated with splanchnoptosis. Godart-Danheiux investigated 871 cases to determine the relationship between enteroptosis and movable kidney, and concluded that there was no parallelism between the two conditions. Enteroptosis and nephroptosis occur at different ages, and under influence of different factors.

Cruveilhier, Senator, and others attribute to tight lacing an important place in the causation of wandering kidney, the constricting bands forcing the kidney downward, elongating its vessels, and producing by pressure absorption of its fatty capsule. It plays its part, no doubt, but we know that the displacement does occur in the male, and in female children before the corset-wearing age. Becker and Lenhoff, with this point in view, examined 24 young Samoan women who had never worn any sort of constricting band about the chest or waist, in 6 of whom the kidney was palpable.

The absorption of fat from the fatty capsule, we are taught by Landau, permits loosening of the peritoneum, and allows the kidney to wander in the subperitoneal space. The fatty capsule has no supportive power. It is probable that in general emaciation, in which absorption of the fatty capsule may take place, there is general weakening of all supports, permitting more or less descensus of the kidney.

Traumatisms, acute or chronic, are assigned causes. When we consider the relations of the kidney and its supports, it is difficult to ascribe any material importance to acutely active traumatism. But if we consider the straining at defecation or urination, a chronic coughing or persistent vomiting, we have in the force applied through the diaphragm a chronically acting traumatism, which gradually forces the kidney down, elongates the cross-fibers of the areolar tissue, and separates the leaves of the perirenal fascia, permitting to the kidney unnatural mobility behind the peritoneum.

That all the causes above considered may contribute to the increased mobility of the kidney may be admitted, yet for the organ to become ectopic other conditions must obtain. As Sulzer and Einhorn claim, there must be individual predispositions, and these are found in the shape and depth of the paravertebral spaces, and the length of the renal pedicle.

Wandering kidney is infrequently met with in obese subjects. Slender women with long and sloping lower ribs of marked inward curve are the type that furnish the most frequent examples of wandering kidney. Harris asserts that the essential cause of movable kidney lies in the particular body-form; the chief characteristic of this body-form is a marked contraction of the lower end of the middle zone, with a diminution in the capacity of this portion of the body. His argument is somewhat vague, though we conclude that the potent cause is the pressure exerted by the superimposed liver, which gradually produces the renal displacement.

Wolkow and Delitzin, from inspection of cadavers, show that the kidneys are attached to the posterior wall in the paravertebral spaces, and that these spaces normally are funnel-shaped. When they are cylindrical and shallow, if there is loss of intra-abdominal pressure, the kidney is easily dislodged.

In enumerating the causes of displacement, we must bear in mind that the renal vessels of the right side are much longer than those of the left. The right renal vessels are only covered by peritoneum and have no other connections, while the left are closely connected with the head of the pancreas by cellular tissue. Landau attaches much consequence to the arrangement of the

renal vessels to account for the greater frequency of mobility of the right kidney over the left.

PATHOLOGY.—When the kidney is loosened from its bed so that the normal respiratory excursion is increased to such a degree as to permit palpation of the entire organ below the costal margin, there are certain morbid changes which may be at once both the cause and effect of the displacement, and to these epiphenomena the symptoms are largely due. Lesser degrees of mobility do not affect the relations of the kidney to its ureter and vessels, nor do they disturb the action, by pressure or traction, of adjacent parts. In descending from its bed deep in the paravertebral recesses, it rises to the level of the vertebral convexity, that is, it approaches nearer the anterior abdominal parietes; the advancing lower pole is arrested by the taut mesocolon, and the force now applied to the upper pole is no longer in the long axis of the kidney, but acts upon the upper half carrying it backward and downward, and serves to bring the organ somewhat into horizontal position with convexity downward. This is the typical attitude of the displacements dependent upon the usual causes, yet the organ may lie in any position. Loose kidneys have no permanent position. Upon exertion, especially in the erect posture, the greatest displacement occurs, with a tendency to spontaneous reduction on assumption of recumbency. The line of descent is downward and forward, so that the organ may be felt in the anterior portion of the lumbar, umbilical, and iliac regions. In extreme cases the kidney may be a permanent resident of the true pelvis.

In the etiology we have placed but little stress upon the fatty capsule as a supporting agent; this structure is usually found attenuated, but we assume that it merely participates in the general emaciation which is frequently present in subjects of wandering kidney. Often, however, it is abundant, hangs loosely about the kidney, forming a part of the retroperitoneal sac in which the kidney moves, or else it is attached to the kidney and moves with it.

The displacement of the kidney necessarily produces traction upon the blood-vessels, which may lead to their constriction, or even, as reported, to thrombosis. The blood-stasis induces venous congestion of the kidney, increasing the size of the organ and

materially disturbing its function. A more important effect is the alteration of the angle of insertion of the ureter into the pelvis of the kidney. The ureter may be constricted or occluded by torsion, or the acute angle of insertion may lead to valvular formation, the transient obstruction causing intermittent hydronephrosis, a subject fully discussed under that caption. To ureteral occlusion and impeded circulation the symptoms of movable kidney are largely due.

It is, perhaps, too emphatically stated that the kidney moves in a subserous sac, without disturbing the overlying peritoneum. That such is the case in moderate degrees of displacement is true; but when the kidney descends to or below the umbilical line the peritoneum is carried in advance, and traction is made upon the reflections to the liver, duodenum and pylorus on the right side. Herbert Bramwell, in recording an autopsy of a case in which there was wandering kidney, says that before anything was removed from the cavity it was distinctly seen how the kidney tightened and dragged upon the peritoneal covering of the pylorus; and further, that the kidney did not glide under the peritoneum, but carried that membrane with it. The effects of this peritoneal traction are to produce duodenal or pyloric obstruction intermittently, and to occlude by displacement the biliary channels. We also notice that the hepatic flexure of the colon is drawn downward. These results may be chargeable to the kidney when not due to general enteroptosis.

I am not in position to define the relation which exists between wandering kidney, diseases of the bile-passages, appendicitis, and uterine retrodisplacement. I have just intimated how traction upon the peritoneum may constrict the bile-ducts. It is a common observation to find mucous enteritis associated with movable kidney. Pressure directly upon the duodenum may be even so great as to give rise to a duodenitis, and from these mechanical and inflammatory causes inflammation of the common bile-duct may develop and initiate either a cholecystitis or cholelithiasis.

A wandering kidney obstructs the venous circulation of the appendix and predisposes to infection, or, by direct pressure, the chronic traumatism may result in appendiceal inflammation; the visceral prolapse may cause kinking of the appendix, inter-

fering with its peristalsis and circulation. While we recognize the occasional coexistence of appendicitis and movable kidney, I have only once been confronted with the necessity of operating for both conditions. Further observations will be required to prove the correctness of Edebohls' statement that 3½ per cent. of all women have both symptom-producing movable kidney and appendicitis.

Retrodisplacement and descent of the uterus, with relaxation of the ligaments, are emphasized by all authors as associate phenomena, either as cause or effect of nephroptosis. We must bear in mind that the rectovesical fascia is but a continuation of the iliac and lumbar fasciæ, and that a process of this, dividing, encases the kidney; its prolongations through the fatty capsule constitute the chief support of the kidney, and we find in this sufficient anatomical reason to attribute to uterine displacements some consequence in the etiology of wandering kidney. Both, however, may be playing a part in general splanchnoptosis.

My clinical experience accords with that of authorities generally, that it is very unusual to meet with movable kidney upon the left side, and still more so to meet with bilateral displacements of the kidney, yet it must be remarked that Edebohls found it necessary to anchor both kidneys in 74 out of 186 patients, about 39 per cent. This proportion appears high; Noble says that only in about one-tenth of his cases are both kidneys displaced.

Symptoms.—We should think of wandering kidney in all women who present themselves for examination in whom the body-form shows a long thorax, narrow waist, and full belly, and in whom there is a deficiency, either recent or long-standing, of adipose tissue, who complain of dyspeptic symptoms, vague pains, and menstrual disorders. It is further suggested by the histories of multiple pregnancies, or of some wasting disease. This is the familiar type, yet it may be met with in the young, robust, athletic man, and occasionally in women of quite different conformation. It cannot be too impressively stated that the kidney may descend its full length from its normal position without exciting symptoms, and the ectopic position may be recognized merely by accident. The complexus of symptoms presented by a vagrant kidney is, in the main, misdirecting; occasionally only are the

symptoms localized to the renal region. The referred disturbances are misleading, and may simulate many intraperitoneal affections. Happily, in the physical examination we have a positive means of diagnosis. A familiarity with the suggestive symptoms leads us to make careful physical investigation. It is all important, therefore, that every symptom, subjective and objective, should receive attention.

Pain: In the course of this affection pain, varying from a mere discomfort and dragging sensation to severe paroxysms, exists. It is not always the most conspicuous symptom, but certainly the most constant. It is influenced by exertion; it not infrequently interrupts muscular effort. It is relieved oftentimes by rest in the recumbent posture. It exacerbates with menstruation, and is intensified by constipation. It is sometimes relieved by pressure or manipulation, or it may become characteristic under pressure. If violent in its onset it would indicate a traumatic cause, with sudden extrusion of the kidney from its bed. It is more chronic, with increasing distress, in all cases due to the usual causes. It is described as a dragging pain in the loin, and referred to the groins and thighs; it is rarely lancinating or reflected upward, but is often referred to the epigastric or umbilical regions. It becomes colicky when the renal vessels become constricted or the ureter contorted. It is attributable to pressure of the kidney in its abnormal location, to traction upon its attachments, and to intracapsular tension. Rafin proved by the use of the ureteral catheter that the painful crises of wandering kidneys are due to retention of urine from kinking of the ureter.

Gastric disturbances are often only such as are common in nervous dyspeptics, but they may assume decided prominence, and may appear so conspicuously in the symptomatology as to divert our attention entirely from the kidney. This gastric irritability, with nausea and vomiting, is attributed by some to mere sympathetic disturbance; by others, to pressure upon the duodenum; but it is probably due to traction upon the peritoneum. Mr. Bramwell reports a case of extreme gastrectasia due to spasmodic pyloric obstruction consequent upon a freely movable kidney. The nephroptosis may be associated with general enteroptosis, in which case gastric symptoms are conspicuous. The gastric crises may occur intermittently with renal obstruction; the vom-

iting is then a distressing and persistent symptom, occurring so early that we can scarcely attribute it to uremia. A sense of nausea and sickness may be developed by pressure upon the displaced organ.

Intestinal Disturbance: Constipation is the rule with patients who are the subjects of this abnormality. Beyea and others assert that a movable kidney often produces a mucous enteritis, a reflected nervous disease through Auerbach's or Meissner's plexus.

Hepatic Disturbances: Occasionally jaundice occurs. We have dwelt in the pathology upon this complication, and reiteration is not demanded further than to discredit the theory of direct compression of the bile-ducts by the kidney, holding rather that it is due to traction upon the duodenal reflection of the peritoneum.

Nervous Phenomena: A perturbed nervous system is a well-recognized state in subjects of wandering kidney. It must be appreciated that renal displacement frequently follows wasting diseases, or occurs in patients who suffer from malnutrition, and the neurasthenia can be attributed entirely to the constitutional depravity; but that it is in others due to the nephroptosis clinical observation establishes. They occasionally complain of something moving or dropping in the abdomen, and in times of acute distress become mentally disturbed. Dizziness, after pain and gastric disturbances, is one of the most common symptoms. Melancholia is often observed. Men are hypochondriacal. Pulsation of the abdominal aorta, cardiac palpitation, insomnia, muscular weakness, a sense of exhaustion unequal to any task, are the varied phenomena.

Menstrual disorders can be explained usually by associate disease and displacements of the internal genitalia, but the particular significance that menstruation has in connection with movable kidney is that the pain and other renal symptoms become accentuated at each menstrual epoch. We scarcely think that Lancereaux's suggestion of congestion of the kidney will obtain, yet we are not prepared to offer a more plausible theory; however, we have observed the exacerbations at menstrual periods.

Urinary Disorders: The chemical character of the urine is not necessarily altered; albumin is occasionally found. Patients who

suffer from nephritis in any of its forms are not unusual subjects of renal displacements, and then the urine would show the nature of the renal lesion. Vesical irritability is excited by constriction of the ureter. The quantity of the urine is variable; when the kidney is incarcerated there may be partial or complete suppression; and this condition, associated with constipation and distressful vomiting, with local pain and perhaps a tumor, offers a diagnostic problem. When oliguria is followed by polyuria, it is a significant symptom of intermitting ureteral obstruction, probably due to wandering kidney. Hematuria occasionally appears.

Physical Signs.—**Inspection:** We must not limit this to the abdominal area. A general view of the abdomen, thorax, and back should be obtained. There is much in form, though we confess to our mathematical inability and a disinclination to indulge our inquisitorial tastes to prove the correctness of Harris's conclusions in the matter of measurements. The hollow flank, flat epigastrium, narrow waist-line, and rather prominent lower abdomen are noticeable. In patients stripped, standing facing you, a right-side asymmetry may be observed, a distinct tumor pouting at the iliac crest, or near the umbilicus; in others the left side may be more prominent because of the gastrectasia. The flabby, relaxed abdominal wall, dotted with *lineæ albicantes*, declares the previous pregnancies and loss of intra-abdominal pressure.

Palpation: Palpation should be employed with the patient in the dorsal position, shoulders slightly elevated and limbs flexed; then in the lateral decubitus; and in the standing position, leaning forward and bearing the weight largely upon the hands resting upon a table of ordinary height (Noble). All patients should be prepared by diet and purgation. Anesthesia is not desirable, as we wish to obtain the assistance of the patient in voluntary inspiration and expiration.

To palpate the kidney the left hand should be placed in the lumbar region close to the costal margin, while the right is applied in front just below the ribs and external to the rectus muscle. The common error is to search for the kidney too far to the outer side. We may, at first, notice some hollowness or want of resistance in the loin, though this is not reliable. With

our hands placed as above described, we may detect all degrees of displacement. The rounded, smooth, elastic organ descends on forced inspiration, and retires on expiration. In our definition we limited wandering kidney to that degree of mobility in which the entire organ is palpable below the ribs; when it descends, the palpating hand may be depressed above the upper pole, and may confine the kidney in its position of displacement, when the organ can be outlined and palpated with the other hand. If this examination is negative, the other postures should be employed, and it may happen that at any one time the kidney will not descend. If, therefore, the suggestive symptoms demand it, we should make several examinations. Low-lying kidneys may be but very slightly influenced by respiration, and may be felt by simple palpation; when they occupy a pelvic position, combined pelvic and abdominal palpation is required for their detection. A displaced kidney is quite movable inward, outward, upward, and backward. It can be made to disappear into the loin, practically a pathognostic sign. Ordinarily they are not easily displaced downward beyond the position they readily descend to. By palpation we determine that they are separate from the liver.

The *costal stigma,* that is, movable tenth rib, is considered by Stillier a strong indication of neurasthenic tendency, a condition common to enteroptosis and movable kidney; Meinert examined 100 neurasthenic, dyspeptic women to determine the value of this sign, and says there is no value in it.

Though not especially sensitive, pressure or squeezing will cause a sensation of faintness, a sickening feeling which may be attended by vomiting. In thin subjects the hilum may be felt, with pulsating renal artery. The appendix should be palpated and the entire abdomen, not forgetting to ascertain the position of each abdominal viscus. Nor is the examination complete that does not include the uterus and its appendages by combined bimanual palpation.

Percussion: Percussion is of but little value in determining renal displacement, though of infinite assistance when combined with inflation, gastric and colonic, in determining the association of enteroptosis. The resonant loin, said to indicate an absence of the kidney, is indecisive; but by percussion over the anterior

surface of the tumor the resonance of overlying intestines simply proves that the tumor is retroperitoneal.

Auscultation: Landau, Cordier, and Futterer report cases confirmatory, and emphasize the value of auscultation in detection of a bruit in the immediate neighborhood of the displaced organ, caused by the torsion of the renal vessel. This sound and the throbbing aorta have led to the diagnosis of aneurism.

DIETL'S CRISES.—We are indebted to Osler for the revival of this symptom-group, first described by Dietl in 1864. In the course of the history of the wandering kidney there often occur acute exacerbations, frequently attributed to some unusual exertion. The pain becomes acute, colicky, paroxysmal in character, felt in the loin, and referred to the umbilical and inguinal regions. There is muscular rigidity, local tenderness; and a swelling of varying proportions develops. There is vesical tenesmus and scanty highly-colored urine, and perhaps a trace of blood may be found. The bowels are constipated, nausea pronounced, vomiting often persistent. The patient is generally depressed, and may show decided shock. There is great thirst, restlessness, mental depression, the pulse feeble and fast, but may be very slow and weak. The duration of these attacks varies; usually they pass off in a few hours, and may recur with varying frequency. A subsidence of the attack is indicated by free flow of urine, a decrease in the swelling, disappearance of tenderness, relaxation of the abdominal rigidity, and arrest of vomiting. Such attacks are ascribed to congestion consequent upon torsion of the renal vessels. While this enters into its production, no doubt, it is now currently attributed to constriction and occlusion of the ureter, with consequent transient hydronephrosis.

DIFFERENTIAL DIAGNOSIS.—The mere recognition of a wandering kidney does not fulfil the requirements of the diagnosis of the case. If we proceed to the surgical treatment without determining the existence of the many complicating conditions so frequently enumerated in this article, only a small percentage of cases will be benefited. There are also many independent morbid conditions which must be differentiated. The acute exacerbations, described as Dietl's crises, are mistaken for appendicitis, biliary colic, intestinal obstruction, and renal calculus.

Appendicitis: It is useless to attach any importance to age, sex, or condition of patient in making this differentiation. The onset may be sudden in both; gastric symptoms may be present in both; constipation is the rule; fever may be present in both or absent in either; the pulse may be normal in both, or equally disturbed; and in certain cases of appendicitis, vesical irritation may appear. Pain, though valuable in its character and location, like all subjective symptoms, must not be relied upon. So practically we are reduced to physical signs for exclusion of appendicitis.

Wandering Kidney.	*Appendicitis.*
Muscular Rigidity.—Involves the loin chiefly.	Right rectus, and muscular plane immediately overlying appendix.
Tenderness.—General, not defined, and less acute; pressure produces faintness and nausea.	Pressure causes distinctly localized sharp pains; pains may also be referred to other portions of the abdomen.
Tumor.—May be present from first, rapidly enlarges.	Does not develop earlier than 48 hours, and slower in growth.
Shape of Tumor.—Reniform or ovoidal.	Ill-defined borders.
Mobility of Tumor.—May be influenced by respiration; movable upon manipulation; may be displaced in any direction, especially upward and backward into the loin.	Is not so influenced. Fixed comparatively; cannot be made to disappear into the loin.
Percussion.—Usually resonant.	Generally dull, may be resonant under light percussion.
Edema.—None.	Often present near iliac crest or spine.
Auscultation.—A bruit may be heard to inner or upper border of tumor.	Negative.
Wandering Kidney.	*Distended Gall-bladder.*
Subjects.—More common in thin, neurotic women under forty years.	More common in fat women over forty years.

Seat of Pain.—In the lumbar, right hypochondriac, umbilical or inguinal regions; thence downward to sacrum, genitals, and thighs.	Pain of biliary calculus is in the epigastric or hypochondriac region; referred to the back and shoulder, almost always upward, rarely downward to sacrum or thighs.
Urine.—May have suppression, polyuria, or ischuria.	Scanty and high-colored.
Seat of Tumor.—Deeply situated; not visible. Easily felt between the hands.	In close apposition with anterior parietes; visible in thin subjects when of moderate size. By bimanual palpation tumor not readily felt with posterior hand.
Movement with Respiration.—May not be influenced by respiration; if grasped, will not ascend with expiration.	Moves with respiration, will ascend with expiration.
Anchorage of Tumor.—No anchorage and no limitation to its movements.	To the liver at the right parasternal line, lower end moving in arc of a circle about this center.
Mobility of Tumor.—May be moved in every direction, and caused to disappear into the loin.	May be moved upward and laterally, rarely backward, never downward.
Shape of Tumor.—Kidney-shaped.	Usually pyriform or oval—may be sausage-shaped.
Resistance of Tumor.—More yielding and elastic.	A thickened gall-bladder filled with stones gives a hard, resistant tumor.
Percussion.—Resonant over some part of its surface, and has a band of resonance between it and the liver-dulness.	Dull, and dulness is continuous with that of the liver.

Inflation of Colon.—May efface entirely the dulness.	Pushes gall-bladder upward, but will not efface its dulness.
Inflation of Stomach.—Causes tumor to disappear entirely.	Moves gall-bladder to the right.
Pressure of Tumor.—Causes faintness or nausea.	Elicits tenderness.
Pyrexia.—Unusual.	Common.
Riedel's Lobe.—No sharp edge felt.	Sharp edge frequently felt across gall-bladder.
Auscultation.—A bruit sometimes audible at inner and upper border of tumor.	Fremitus may occasionally be felt and heard.
Disappearance of Tumor.—If there is hydronephrosis, disappearance marked by copious discharge of urine.	May disappear without extra flow of bile.

Intestinal Obstruction: Fecal impaction in form and symptoms may simulate a renal tumor. Thorough purgation before examination would disclose the true nature of the mass.

A wandering kidney, by irritation rather than by direct pressure, may simulate or actually induce intestinal obstruction. The acute exacerbations, which sometimes develop in nephroptosis, while usually marked by urinary disturbances, sometimes produce such gastric and intestinal perversion that the diagnosis becomes difficult. The chief point of distinction between mechanical intestinal obstruction and wandering kidney is the absolute arrest of both gases and feces in the one, which is only relatively so in the other. The mobility and shape of the tumor, the oliguria and vesical tenesmus, and the lesser degree of shock, distinguish the wandering kidney.

Renal Colic: A crisis in the course of movable kidney may resemble renal colic. Hematuria is very unusual in movable kidney; a microscopical quantity of blood is a frequent occurrence in calculus. Pyuria is also found in calculus. A tumor is unusual in calculus-disease unless suppurative pyelitis is present, and then its gradual formation and septic symptoms indicate its nature; and if such tumor is formed, it is usually fixed and does

not enjoy the same painless mobility so characteristic of wandering kidney.

Pyloric Obstruction: Allusion has been made to pyloric obstruction and gastrectasia met with in vagrant kidney. A pyloric tumor may be a very movable mass, and may be found quite low in the abdomen. The scanty urine, thirst, vomiting, together with the mobility of the tumor, are features in common with wandering kidney. The history, the age of the patient, rapid emaciation and cachexia, hematemesis, absence of hydrochloric acid, are points in differentiation. Furthermore, a pyloric tumor is usually stony-hard, rests in close apposition to the parietes, is dull upon percussion, and cannot be displaced beyond the reach of palpation.

Hepatic tumors, constricted or somewhat pedunculated, a very rare affection, are usually superficial, their continuity with the liver can be established, they move freely with respiration, which movement cannot be restricted, and are not marked by distinct crises.

Wandering spleen is an oblong, flat tumor, with sharp, notched edge. It is superficial in location, never underlies the colon, and is frequently attended by characteristic blood-changes. Percussion will reveal its absence from its normal position, and an area of resonance posterior and to the outer side of the tumor. These are characteristically diagnostic features.

Intestinal cancer, especially of the cecum, may, on superficial examination, be taken for movable kidney. Such tumors are usually dull upon percussion, are mobile in a very limited area, produce alternating diarrhea and constipation, cachexia develops early, and their relations can be established by insufflation of the bowel.

Uterine and ovarian tumors, when small and pedunculated, may rise out of the pelvis, and may assume any of the positions occupied by wandering kidney. Their method of growth, the absence of tenderness or pronounced pain, restriction to upward displacement, their connection with the seat of origin, and further, their superficial location and percussion-dulness mark them.

PROGNOSIS.—A freely wandering kidney may cause but little disturbance in some cases. In others, health is impaired by the func-

tional disturbances and neurasthenic state to which the patient is reduced. Life is imperilled only when complications supervene. We hesitate to give an operative prognosis. The benefits obtained by Morris, Noble, Edebohls, and others far excel the author's experience in realizing absolute restoration of the patient to health. My mortality is nil, fixation of kidney is permanent, but several patients have remained dyspeptic, anemic, nervous, and readily find other troubles.

Surgical Indications.—The necessity for operation cannot be judged by the degree of mobility of the kidney, though within certain limits it can be said that the more mobile the kidney, the greater the likelihood of its undergoing pathological changes and its giving rise to symptoms. The only symptoms that we can hope to relieve by fixation of the kidney are those due to its mobility, and we should use extreme care in the selection of cases for operation. Dietl's crises always, and even less pronounced attacks, in fact any discomfort referred to the region of the kidney, and any disturbance of the urinary function can be safely attributed to wandering kidney when it is present, particularly so when there is no other demonstrable lesion of the urinary tract. Nervous and digestive symptoms are too frequently the result of an associated pathology for us to promise relief from nephropexy. The surgeon who does so will occasionally get gratifying results, but there can be no accuracy in selecting among several the cause of reflex symptoms, and he is taking desperate chances in failing to make good his promise.

In this last class of cases when operation is done at all it is very essential not only to fix the kidney, but as far as possible to correct all associated pathology in the way of cervical and perineal lacerations, diseases of the uterus and its appendages, and to remove the appendix.

For fixation of the kidney the technic of Edebohls is most satisfactory. The decapsulation of the kidney is a distinct help in relieving any congestive or inflammatory disturbance that has resulted from its mobility, and fixation through the medium of the true capsule without passing sutures into the parenchyma of the organ is a point that should be observed, no matter how the technic is otherwise varied.

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HYDRONEPHROSIS.

DEFINITION.—A distention of the kidney with non-infected fluid, caused by an obstruction to the flow of urine.

SYNONYMS.—Aseptic cystonephrosis, nephydrosis.

FREQUENCY.—In computing the frequency of hydronephrosis we must draw the distinction between clinically recognized cases and post mortem findings. Morris's investigations show that in fully 5 per cent. of necropsies hydronephrosis is found in "marked" degree, yet about 90 per cent. of these cases owe their origin to uterine and other pelvic tumors, whose obtrusive symptoms overshadow the renal retention, and the hydronephrosis is overlooked or disregarded. Osler had seen only 4 cases of intermittent hydronephrosis prior to 1894. Morris tabulates 9 cases upon which he has operated. The writer has encountered two cases of unilateral hydronephrosis from uterine carcinoma, and several others from various causes; and his experience would indicate that it is not an infrequent form of abdominal tumor.

ETIOLOGY.—**Age**: Hydronephrosis may affect the fetus in utero, or may develop from congenital or acquired causes at any period during life.

Sex: For etiological reasons hydronephrosis is more common in women than in men, perhaps in the ratio of three or four to one.

The causes that give rise to hydronephrosis may be either congenital or acquired. Mr. Morris draws a nice distinction between congenital hydronephrosis and hydronephrosis of congenital origin. In the former the hydronephrotic condition exists at birth, while in the latter, though the cause is congenital, many years may elapse before the development of retention and cystic accumulation. An occlusion of any portion of the urinary tract from the calices of the kidney to the meatus urinarius may result in obstruction to the flow of urine and its retention within the kidney. One-third of all cases are accredited with being of congenital origin. It is probable that the proportion is even greater

than this; only in late years has attention been directed to abnormalities in the mechanical arrangement of the ureteropelvic junction. Phimosis, partial or complete atresia of the urethra, folds of mucous membrane at the vesical orifice, are congenital conditions which give rise to bilateral hydronephrosis; if complete, the infant is still-born or dies a few days after birth. Congenital conditions affecting the bladder rarely give rise to hydronephrosis. Malformations of the ureter, valve-like pelvic insertion, kinks and twists, and in some cases an entire absence of the ureter, a defect usually associated with some other malformations, are the most commonly found congenital causes. Roberts refers to two cases of obstruction from compression of the ureter due to an anomalous branch of the renal artery; Coats also reports such a case.

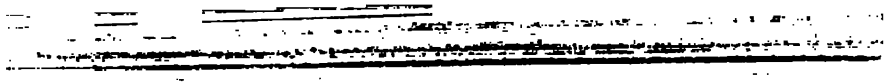
Of 32 cases observed by Roberts, acquired hydronephrosis was found to be due to impacted calculus in 11 instances. This observation appears to have promulgated the belief that renal calculus is a common cause of hydronephrosis; Tillmanns, and other equally high authorities regard it the most frequent cause. Of 142 cases analyzed by Mr. Morris, vesical calculus is given as a cause in 3 cases; there is no instance of ureteral impaction. Of 665 cases of hydrenophrosis collected by Newman, there were 68 due to renal calculi. Ureteral occlusion by calculi, though a common cause of pyonephrosis, should be regarded as only occasionally productive of hydronephrosis.

Strictures of the urethra and prostatic obstruction are the common causes of hydronephrosis in males; in some instances there is vesical retention with damming back of urine through the ureters, but the more usual condition is a consecutive hypertrophy of the bladder-wall, which in itself may prove obstructive to one or both ureters (James).

Malignant growths of the uterus, vagina, bladder, and rectum are regarded by Morris as producing 90 per cent. of the cases of acquired hydronephrosis; they constitute almost one-third of all causes in Newman's table. R. Williams in 78 autopsies, patients dying of cancer of the uterus, met with hydronephrosis in 67 cases. Of 9 cases observed by Cullen at Johns Hopkins, the kidney was affected in all, and in 6 it was hydronephrotic. The ureters may be compressed or displaced, but the usual obstruct-

KIDNEY. HYDRONEPHROSIS.

Organ incised through long axis. The pelvis and calices are dilated, cortex compressed and atrophied and the organ about double the size of the normal kidney. The oblique insertion of the ureter producing valvular obstruction is also shown. The surface of this kidney is shown in Plate XII.



ing cause is in the wall of the bladder. The hydronephrotic distention is very gradual, may be bilateral or single, and often escapes observation.

Simple inflammatory strictures of the ureter are rare causes of hydronephrosis. In 1891 Watson could collect only 6 undoubted cases of hydro- and pyonephrosis so caused, all necropsical. Within the last decade perhaps as many more have been recorded, with operations for the relief of the obstruction, notably the cases of Küster, Fenger, and Kelly.

Movable kidney is the conspicuous cause of intermittent hydronephrosis in the female; it is often accompanied by an abnormal insertion of the ureter into the renal pelvis; it is difficult to say whether the relation of the ureter to the pelvis is the cause or the effect of the hydronephrosis. Of 66 cases of acquired intermittent hydronephrosis movable kidney existed in 28 (Terrier and Boudouin). As taught by Landau, the repeated displacements, twisting and kinking of the ureter are sufficient causes for renal retention.

Extreme conditions of hydronephrosis may result from obstruction offered by insertion of the ureter into the renal pelvis at an acute angle. Fenger contributes a historical *résumé* of all cases of hydronephrosis operatively treated with a view to overcoming the cause of the obstruction. There are about 30 cases considered, and in 26 of these there was a valve-formation of the ureter, with oblique insertion into the dilated pelvis, or a stricture of the ureter, with or without abnormal insertion into the pelvis.

Other causes which are only occasionally active in giving rise to hydronephrosis are traumatism, abdominal tumors, pregnancy, uterine displacements, and fecal impaction. Bland Sutton says that one of the most startling facts connected with his study of hydronephrosis is that in many of the larger cysts no obstruction could be demonstrated, and the clinical histories failed to throw any light upon the causes producing the condition; yet in by far the greater number of cases the obstruction is easily demonstrable.

PATHOLOGY.—Hydronephrosis may affect one or both kidneys. In the unilateral retention the obstructing cause is in the ureter or pelvis of the kidney; exceptionally only does it proceed from

causes in the true pelvis, yet in neoplasms of the bladder, primary or secondary, only one ureteral orifice may be encroached upon. Bilateral distention of the kidneys usually arises from vesical or prevesical obstructing causes, or from symmetrical congenital abnormalities. The resulting tumor may be either constant or intermittent; its permanency depends upon the completeness and constancy of the obstruction.

The changes which take place in the kidney in consequence of an interruption to the outflow of urine are governed by the manner in which the obstructing cause acts. A *sudden, complete* and *permanent* occlusion of the ureter results in such rapid atrophy of the kidney-substance that the consequent distention, though present, is usually small. The pressure of the retained urine within the uriniferous tubules and capsules of Bowman is greater than the vascular tension in the glomeruli, and the secretion of urine is arrested. The primary enlargement consequent upon complete obstruction is, in a measure, due to the retained urine, but Albarran thinks that the kidney enlarges at least one third in size from vascular congestion. The rapidly resulting atrophy in such cases is attributable both to pressure of the retained fluid and to degeneration, the result of congestion. The vessels may become thrombosed or obliterated by thickening of their walls and formation of fibrous cords, the tubules are compressed, and finally the whole parenchyma of the kidney is converted into connective tissue. In the acute form the fluid may force the capsule away from the gland-structure of the kidney, which is then found collapsed and atrophied in the bottom of a large sac formed by the capsule of the kidney, as in the unique case reported by Marsh. These changes can only take place when the other kidney hypertrophies and assumes the burden of both organs.

If the obstruction is *complete* and *temporary,* the intrarenal pressure is relieved with the release of the obstruction, and there is an abundant flow of urine, which is due to the congestion and to the diminished resistance offered to exosmosis, and not so much to the renal retention of urine. If this condition of distention and relaxation is *recurrent,* there is gradual atrophy of kidney-substance and progressive distention of the pelvis and calices, the papillæ are flattened, the kidney's retention-capacity

KIDNEY. HYDRONEPHROSIS.

The surface of the organ shows the irregular lobulation resulting from dilatation of the calices. The dilated pelvis and ureter, above point of obstruction, are well shown.

The incised surface of this specimen is shown in Plate XI

is increased, and the pressure of the retained urine compresses the cortical substance against the capsule. If the obstruction becomes permanent the growth of the hydronephrosis will continue as long as there is any secreting substance left; and even after all glandular structure is destroyed, the secretion from the mucous membrane may add to the accumulation.

In *complete* obstruction, either sudden or gradual and progressive, the hindrance to the outflow of urine is sufficient to bring about the same intrarenal changes as above described, yet it is accomplished in a most gradual and insidious manner. Mr. Morris contends that such obstructions as permit leakage usually result in renal atrophy without the formation of an appreciable hydronephrosis. Stricture of the ureter may be taken as a type of this form of obstruction, and the instances quoted in the etiology conclusively show that an incomplete obstruction, which does not clinically intermit, will cause large hydronephrosis. Practically all hydronephroses which form tumors intermit, but authorities would limit the phrase "intermitting" hydronephrosis to those cases in which there is a great diminution in or actual disappearance of the tumor.

The size of the hydronephrosis depends largely upon the completeness and acuity of the obstruction. A sudden plugging of the ureter may produce a hydronephrosis but little larger than the actual size of the kidney, the atrophied renal structure giving place to the moderate accumulation. If the cause is incomplete or intermittent and gradual in its action, the resulting accumulation may form a tumor of enormous dimensions. No article upon hydronephrosis is complete that does not refer to the almost incredible case reported by Mr. Glass, in which the hydronephrotic sac contained thirty gallons.

The morphology of the swelling is subject to much variation. When the obstruction is in a calyx, sacculation of one portion of the kidney may occur. A ureteral obstruction usually leads to uniform distention of the renal pelvis, with gradual encroachment upon the kidney-substance. Such swellings retain the kidney-shape until there is complete atrophy. This behavior is by no means uniform; the distention may be confined to the pelvis and infundibulum. Isolated retention-cysts may be found in any

part of the tubular structure of the kidney. The dilatation of the pelvis may be uniform (Morris), or asymmetrical (Mynter).

The irregular evolution of the sac distorts the relation of the ureterorenal junction, which in itself may become an additional cause of obstruction. If there is a congenital abnormality in the relation of the ureter to the renal pelvis, it is apparent that, if from any transient cause urinary retention should occur, sacculation of the pelvis with valvular obstruction of the ureter is a natural consequence. In advanced cases of hydronephrosis which involve the pelvis and calices, the whole organ is converted into a cyst or a number of cysts with fibrous septa. The cortex is compressed and with the capsule forms the limiting wall of varying density and thickness which may undergo calcareous degeneration. Its outer surface is irregular and acquires adhesions to surrounding structures, and the inflammation extending through the peritoneum, it even acquires attachments to the abdominal viscera, into which the sac may rupture. This complication is usually followed by infection of the sac, and if the fistulous communication should close, any subsequent accumulation is likely to be purulent. Rupture into the peritoneum generally gives rise to peritonitis, but the aseptic fluid may be absorbed, and in this way spontaneous cure has been known to occur. A frequent and greatly to be feared complication which may befall a hydronephrosis is infection of the sac and contents. The conversion of a hydronephrosis into a secondary pyonephrosis is the work entirely of pyogenic microörganisms. Elsewhere we have referred to experimenters' denial that infection could occur along the channel of the ureter. Clinical evidence contradicts this conclusion of the laboratory-workers, and there is no doubt but that infection of open or intermitting hydronephroses may occur by this route; yet the close relation that the sac occupies to the bowel-wall and the lowered resistance of the tissues permit infection without fistulous communication. The softening and disintegration of the sac in consequence of infection render it very liable to rupture, either internally with fatal effect from peritonitis, or the pus may escape extraperitoneally and become perinephric.

The contents of the hydronephrotic sac is urine secreted against pressure; it therefore resembles very closely the serum

of the blood. The greater the renal atrophy the more closely the fluid partakes of the character of the normal serum, and the less the urates and uric acid. The specific gravity is usually low, 1,006. The fluid is albuminous, slightly alkaline or neutral. The color may be altered by an admixture with blood; its turbidity is due to mucus, phosphates, and epithelial cells. In old cases the contents may be of a colloid nature.

The site of the obstruction determines the associate changes in the ureter. The duct above the obstruction is distended, and may attain to the size of a small intestine. Double hydro-ureter is a rather common autopsical finding from congenital occlusion, and from contraction due to malignant infiltration of the bladder or pelvic tissues. If the obstruction is located in the course of the ureter, the distal end will be relaxed and atrophied from disuse.

SYMPTOMS.—Hydronephrosis is a complication or reflex effect of an antecedent disease or abnormality, the symptoms of which may be expressed alone by the development of the complication; and, in turn, the behavior of the hydronephrosis is influenced largely by the causative condition. The symptoms of the numerous and varied causes enter into the history of hydronephrosis, and in this we encounter the unrecognizable congenital occlusions, or the gradual obstruction consequent upon cancerous infiltration of the pelvic organs; in other instances the storm of nephritic colic from calculus may mark the beginning; and very commonly we are able to obtain the history so characteristic of movable kidney with its intermitting crises. In consideration of the various causes and their effects we have attempted to show that hydronephrosis may exist without an appreciable tumor, and in this concealed form it may in the most subtle manner cause renal atrophy and terminate fatally. Under other circumstances there may be a tumor of moderate or enormous dimensions, of stable or constant existence. And under certain mechanical arrangements the tumor is intermitting, partially or entirely disappearing to reform at irregular intervals. It seems well, therefore, to consider the symptomatology of hydronephrosis in its three clinical aspects.

1. **Tumor Absent**: Hydronephrosis may exist without giving rise to a recognizable enlargement from two causes: a sudden,

complete and permanent occlusion of the ureter; and from a gradual occlusion which permits some leakage. Calculus-obstruction may be taken as the type of the former, and cancerous involvement of the latter; yet many other causes may act in a similar manner.

In acute obstruction there is an exaggeration of the "ureteral beats." It must be understood that this duct is not a passive channel, but that it acts like the esophagus in propelling its contents downward. Therefore, any interference with its rhythmical impulses is resented, and there is an exaggerated beat, which by nervous connection is transmitted to the bladder. Frequent micturition is the effect of this muscular excitation, and is one of the earliest and most important symptoms indicative of interference with the free action of the ureter, whether it proceeds from causes within or without. The amount of urine voided is decidedly less than normal, and becomes of high specific gravity as it represents the product of the uninvolved kidney which has assumed double work. In all suddenly acting causes which completely occlude the ureter pain is a conspicuous symptom. It is expressed at first as nephritic colic, but the cause still persisting, the pain loses its paroxysmal character for simply a dull ache in the lumbar region, with radiations characteristic of renal troubles. In these acute cases there are always marked digestive disturbances; constipation and tympany may be present; nausea is a constant accompaniment, and vomiting a common occurrence. The skin is dry, thirst usually pronounced, and the further manifestations depend solely upon the capacity of the other kidney to take on the work of both. If there is previous disease of the opposite kidney, or if there should happen to be a simultaneous occlusion of both ureters, as sometimes occurs in calculous conditions, anuria develops; the patient may die quickly from uremic poisoning, or may pass through a few days of tolerance to die finally of uremia.

The *physical signs* in unilateral hydronephrosis from sudden occlusion, in which no tumor is formed, are few and not very characteristic. Unfortunately, care is not taken to ascertain those that exist in the early stage. While there is no tumor, yet, as we have shown in the pathology, there is necessarily an increase in the kidney's bulk of about one-third. In any given case it is

impossible to appreciate this unless the clinician is familiar with the size of the organ before the occlusion. Marked tenderness in front and behind is always to be noted and indicates the renal tension. The urine may contain blood; especially is this true in calculus-obstruction, and is sometimes noticed in the torsion from dislocation of the kidney. Cystoscopic examination when practicable, aided if necessary by ureteral catheterization, will furnish conclusive evidence of the arrested renal urine on the side involved. The ureteral bougie may locate the site of the obstruction.

An impediment, but not absolute obstruction, to the outflow of urine may be located in any portion of the urinary tract. If its seat is in the pelvic organs the resulting distention will generally be bilateral, though frequently of unequal degree; if in the ureter or pelvis of the kidney, then only one kidney is involved. The anamnesis of the consecutive hydro-ureter and hydronephrosis would be found in the symptoms of the obstructing disease, whether it be prostatic obstruction, vesical neoplasms, primary or secondary, or ureteral stricture. As before suggested, the most common condition giving rise to partial occlusion is cancer of the cervix uteri. In all these states the primary trouble has most active symptoms, and but little attention is given to frequent micturition, oliguria, lumbar pains, dry skin and thirst, stupor deepening into coma, delirium and convulsions, conditions that denote the rapid renal atrophy. The leakage is sufficient to prevent the development of a palpable tumor, but the autopsy will reveal a moderate accumulation of fluid in the pelvis of the kidney, with great thinning and atrophy of the renal substance; and if the obstruction is in the lower end of the ureter or beyond, these ducts are found irregularly dilated and tortuous.

2. **Tumor Constant**: When the renal retention is due to complete obstruction, or, if incomplete, the leakage is not sufficient to cause any appreciable alteration in the amount of accumulation, the resulting tumor slowly enlarges as long as it is supplied with fluid from the renal structure or mucous membrane. Information may be obtained of some acutely acting cause, or the distention may have developed insidiously without any recognizable obstructing condition. It is usually chronic in its course; for, while the tumor may be recent in development, it

must necessarily arise from a chronically acting cause. It is characterized by subjective symptoms which vary with almost each individual case. Frequent micturition but a diminution in the normal daily amount of urine are symptoms that may be noticed by the patient. There is impairment of the general health as indicated by loss of flesh, the skin is dry, and digestive disturbances are complained of. Though not a cause of acute suffering, a stable hydronephrosis generally gives rise to pain in the lumbar region and side affected. Exercise, manipulation, and constipation add greatly to the discomfort.

The tumor in its physical characteristics depends upon its size. A moderate distention of the kidney may retain the contour of the organ, if the distention is within the hilum; but when the renal pelvis is alone distended, equally or asymmetrically, the tumor may be of any shape. It usually lies well in the loin, fills the costo-iliac space, pushes the liver upward, and evolutes downward and toward the median line. It is not, as a rule, tender upon pressure, is elastic and resisting under palpation, and fluctuation is encountered when the sac-wall is very thin or when leakage lessens the tension. It is dull upon percussion on its posterior and external aspects. As the tumor grows, the colon is generally pushed in front of it, and the intestines displaced downward and to the opposite side, so that across the inner and anterior surfaces there is a line of resonance over the path of the colon; this relation is by no means constant, for the colon may pass along the lower border, or may even be displaced to the outer side; this last relation is not at all uncommon in very large tumors, and with this position of the colon the intestines usually lie in front of the tumor. The insufflation of the colon with gas, and auscultatory percussion will define the relation of the gut to the tumor. In permanent hydronephroses the cystoscopic examination will show urine from only one ureter. The obstructing cause will occasionally permit the introduction of a ureteral catheter, and of the withdrawal of fluid directly from the kidney. If such a test can be successfully employed, it would be conclusively diagnostic.

3. **Tumor Intermitting:** There are certain causes which temporarily obstruct the ureter, producing moderate hydronephrosis at first. The repetition of this distention and relaxation results

in such atrophy and dilatation that a palpable, in some instances a perceptible, tumor will form with each reoccurrence of the occlusion, and will disappear with release of the constriction. Such intermitting hydronephroses are especially common as phenomena in movable kidney, and in those congenital conditions in which the ureteropelvic angle of insertion is very acute. The symptoms which accompany this form of renal retention are very pronounced and diagnostic. Not infrequently the patient has become aware of the existence of a movable kidney, and for a variable time may have suffered from the manifold disturbances incident to its changing relations. The hydronephrosis rarely appears as the first symptom of complaint. There are attacks of pain in the side, renal in location, often very severe and in character closely resembling a seizure of the colic of renal calculus. This resemblance grows closer when, in the attack, a little blood is noticed in the urine, which exceptionally occurs. The great desire to micturate adds to the distress, and oliguria or even absolute suppression for a time may be present. Complete anuria would argue that there was a single kidney, or else that the opposite organ was in some way crippled. Intermitting hydronephrosis, as far as clinical evidence goes, is almost always a unilateral affection, and most frequently on the right side; yet Mr. Sutton enjoyed the rare opportunity of witnessing a double hydronephrosis, with the disappearance of one tumor during a celiotomy for a uterine fibroid. Returning to the symptom pain, its occurrence denotes that complete occlusion of the ureter has taken place, and the pelvis is distended with retained fluid, while the entire organ itself is in a state of congestion. In the most acute cases the patient manifests great restlessness, both mental and physical; there is marked depression, pulse is fast and weak; tympany, constipation, and vomiting are present. The duration of the attack may be only several hours, or, the symptoms becoming modified, it may last through days; with relief of the obstruction, there is sudden and marked polyuria and subsidence of all symptoms.

Other cases of intermitting hydronephrosis pursue a very different course. Silently and gradually the tumor will grow to a certain size, give rise to but little distress, and unexpectedly surprise both physician and patient by suddenly disappearing, its

disappearance being marked by a copious discharge of urine. This disappearance of an abdominal tumor attended by an abundant flow of clear urine is regarded as almost pathognomonic of a relieved hydronephrosis, yet it must not be forgotten that a copious discharge of urine will follow the rupture of other abdominal cysts, especially ovarian, into the peritoneal cavity, their contents being absorbed and eliminated by the kidneys. Thomas Wilson, of Birmingham, England, has collected operative and post mortem proof, showing very clearly that the disappearance of an abdominal tumor followed by polyuria is no more characteristic of hydronephrosis than it is of ruptured ovarian cyst.

When the ureter is blocked by a stricture or other cause in its distal or vesical end, hydronephrosis may occur without great distention of the ureter; or the reverse may occur, there may be a large accumulation of fluid in the dilated ureter, hydro-ureter, without renal involvement. The symptoms of hydro-ureter are not distinctive. Kelly refers to two cases in which there was a sacciform dilatation of the ureter which could be easily palpated through the vagina, and in one instance was mistaken for a vaginal cyst. When greatly distended the ureter may be palpated in favorable subjects. The catheterization of the ureter, and the escape of a continuous flow of a clear fluid as soon as the instrument passes the obstruction and before it reaches the pelvis of the kidney, would indicate hydro-ureter; but it does not exclude hydronephrosis.

Differential Diagnosis.—**Pyonephrosis:** It is often impossible to differentiate hydronephrosis from pyonephrosis. This is especially true in those cases which present themselves bearing a small tumor and with an indefinite or obscure history. If, however, we can obtain the information as to the exciting cause, we are guided in our conclusions. The history of infection from the lower urinary tract, or the symptoms common to renal calculus, and occasionally the development of the renal trouble after some of the acute exanthematous fevers, would point to the accumulation as purulent. If to this knowledge we add the symptoms of sepsis, with pain and local tenderness, the presence of pus may be strongly suspected. In pyonephrosis the urine generally contains pus sometimes before there is complete occlusion, and if

there is not absolute obstruction, the urine from that kidney will show the presence of pus.

A **perinephric abscess** may be incident to primary renal suppuration, or secondary to infection elsewhere. The tumor formed is fixed and immovable, the muscles are rigid, the skin is often discolored and edematous, pain and tenderness are marked, there is no necessary variation in the character or quantity of urine, and the constitutional symptoms of irregular fever, with distinct chills or chilly sensations and sweating, denote the septic process.

Cysts of the liver and spleen are usually hydatid in nature; they bulge into the abdomen or press upward against the diaphragm; their continuity with the organ involved can be established; urinary, vesical, and ureteral examination will exclude involvement of the kidney.

Ovarian Cyst: A large hydronephrotic cyst may be mistaken for an ovarian cyst; the error has occurred repeatedly. A careful investigation into the history of the case, ascertaining the probability of urinary obstruction, should be the first step in differentiation. The seat and manner of growth should be determined. Ovarian tumors grow from below upwards, usually commence upon one side, then become central, are in close relation to the anterior parietes, and push the intestines above and to the sides. Ovarian tumors are usually painless in their early history. They do not affect the character of the urine, though they disturb the function of the bladder. Their connection can usually be determined. They are dull over the surface, resonant in the flanks, and capped by a crown of resonance.

Distended Gall-Bladder: The points in common in these affections are numerous; especially confusing is the fact that they both may follow a severe attack of pain, the nature of which cannot be definitely determined. The gall-bladder may be found quite out of its normal position, occupying the position in which we would look for hydronephrosis, especially when associated with movable kidney. Gastric and intestinal disturbances are common to both, and the tumor of either may suddenly disappear. A gall-bladder moves with respiration; its continuity with the liver can be established by palpation and percussion; it is generally dull upon percussion; the colon lies to the outer side;

it is usually in close relation to the abdominal wall, and does not bulge into the flank. There is no oliguria, and cystoscopic examination is negative.

Prognosis.—The dangers that attend hydronephrosis are: first, those that proceed from the cause producing it; second, infection of the cyst and contents; third, the development in the other kidney of some acute trouble which may precipitate suppression of urine; fourth, rupture with peritonitis; fifth, the pressure-effects of large accumulations upon other viscera.

Surgical Indications.—The first step in the management of hydronephrosis is to determine the urinary sufficiency of the opposite kidney. And we must further bear in mind that, although a hydronephrosis of magnitude may exist, the compressed and seemingly atrophied renal structure may possess a secreting power sufficient to conserve life and health; this was notable in Küster's and in Morris's cases.

There are two aims which we should keep in view in the treatment of hydronephrosis: first, remove the cause; second, preserve what is left of the kidney if possible. *Nephrectomy* is therefore reserved for those hydronephroses which have become infected or in which there is no kidney-structure left. Such cases are not cured by aspiration; and *nephrotomy* has a mortality of nearly 50 per cent., according to Rovsing, and in the remaining cases is often followed by an unpleasant fistula.

In those hydronephroses secondary to malignant infiltration about the ureters all operative considerations are futile; even palliative nephrotomy for the relief of anuria can give the patient but small comfort. Such needless procedures discredit surgery.

Obstructing calculi should be removed, uterine displacements corrected, and pelvic tumors ablated.

We are especially indebted to Küster, Fenger, Mynter, and Briddon for the method of management of those cases due to stricture of the ureter, and to valvular obstruction from abnormal insertion into the renal pelvis. In a most excellent article by Fenger in which he reviews all the operations, about 30 in number, undertaken with this conservative idea, the following conclusions are reached; extrapelvic plastic operation, that is, longitudinal division of stricture and valve, with transverse closure, as suggested by Fenger, is the operation of choice; it has been performed 11

times with 10 good functional results and one failure. The transpelvic plastic operation may be the operation of necessity in large sacs, because of the difficulty in reaching the ureter outside of the pelvis. Resection of the strictured ureter with reimplantation into the renal pelvis, the ureteropyeloneostomy of Küster, has been done 6 times, successfully in 3, abandoned for nephrectomy in 2, one terminating fatally from sepsis. When the renal pelvis is redundant, the excess may be excised with closure of the wound by sutures, the *capitonnage* of Albarran; or the pelvic wall may be shortened by infolding and uniting the folds by suture, the *pyelo-plication* of Israel. Of the 30 cases treated by these several methods, 22 were functionally effective. Modern surgery is indebted to Fenger for his original work in this line, but more especially for the elaboration of the subject and the setting-forth of the surgical indications to meet individual conditions.

Ureteral stricture may be amenable to dilatation. From Dr. Kelly's experience one would judge that it would require a deal of time and patience. Ureteral stricture near the vesical end, if not susceptible of dilatation, should be treated by resection of the ureter with reimplantation into the bladder. When the stricture occurs in the course of the ureter, it should be treated after the method of Fenger, longitudinal incision with transverse closure, when practicable; but where the ureter is reduced to a mere fibrous cord, the strictured portion should be excised, and the channel reëstablished by Van Hook's method of lateral implantation.

Transient or incipient hydronephroses are frequently encountered in movable kidney. If left to pursue their course they will develop large tumors. *Nephropexy* is, therefore, a preventive measure, and may, without the aid of any further procedure, overcome a developing hydronephrosis.

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RENAL SUPPURATIONS.

DEFINITION.—Bacterial infection of the kidney, resulting in pus-formation. We embrace under this general head all suppurative conditions affecting the kidney or its pelvis, anatomically designated *suppurative pyelitis, pyonephrosis, suppurative pyelonephritis,* and *renal abscess.*

Suppurative pyelitis is a purulent inflammation of the mucous membrane of the pelvis of the kidney.

Pyonephrosis is an accumulation of pus or purulent fluid in the pelvis of the kidney without secondary infective foci in the parenchyma of the organ.

Suppurative pyelonephritis is a simultaneous purulent inflammation of the kidney and its pelvis, erroneously and objectionably styled 'surgical kidney.'

Renal abscess is a suppurative nephritis, primarily interstitial, and not preceded by pyelitis or pyelonephritis.

FREQUENCY.—Renal suppuration in its several forms appears conspicuously in mortuary reports, but the imperfect examinations and loose nomenclature make it impossible to determine the relative frequency of the several conditions. In the great majority of cases the renal pelvis is first invaded. Of 270 cases dying of urinary disorders, Goodhart found that 130 suffered from suppurative pyelonephritis. In ten years at the Middlesex Hospital, Mr. Morris found 26 cases of suppurative pyelonephritis, and 12 cases of pyonephrosis.

ETIOLOGY.—The essential factor in all forms of renal suppuration is the action of pyogenic organisms. As we have so often previously stated, the mere presence of germs in the kidney is not sufficient to give rise to suppuration; there must be local inviting conditions which favor their retention, culture, and destructive action. These conditions may arise from constitutional causes exceptionally, occasionally are the result of direct traumatism, but in the vast majority of cases the state of lowered resistance is created by renal congestion consequent upon interference

with the free discharge of urine; the retention of that fluid furnishes an excellent culture-medium for germ-growth.

Schmidt and Aschoff report 14 cases of pyelonephritis due to the bacillus coli communis, and from experimentation conclude that the changes found in the kidneys of rabbits after the injection of the bacillus coli communis into a ligated ureter correspond to those seen in suppurative pyelonephritis in man. Albarran and Cottel, in 43 cases of suppuration in and around the urinary tract, found anaërobic bacteria, with or without aërobes, the former leading to general peritonitis or secondary infections elsewhere. Sternberg regards ascending infections involving the kidney as generally due to the bacillus coli communis and says that up to date but two species of bacteria have been demonstrated to be the cause of pyelonephritis; if others are found their presence is accidental, and it is probably a mixed infection. Renal urine containing pus is usually acid, and, as the above author says, the point that we must remember is that the colon-bacillus produces acid urine. Alkaline urine, due to the decomposition of urea, results from the action of the bacillus proteus vulgaris, sometimes from a staphylococcus; this statement is clinically verified by Brown's careful investigations. Of 5 cases of calculous pyuria, in which the urine obtained by ureteral catheterization was alkaline, Brown found the bacillus proteus vulgaris in three, and a white staphylococcus in two. In a specimen of acid urine the bacillus coli communis was found in pure culture.

To comprehend well the channels by which infection may reach the kidney we must look into the antecedent conditions which act as contributing causes. Inflammations of the lower urinary tract, whether obstructive or not, occupy the most conspicuous place in the production of pyelitis, suppurative pyelonephritis, and pyonephrosis. These inflammations are more common in young adults and in those of middle age of both sexes, and are often of gonorrheal origin; yet this specific germ, the gonococcus, only appears in about 3 per cent. of cases as the pathogenic agent in renal suppuration. The common post mortem findings in suppurative conditions of the kidney are lesions inflammatory in nature, obstructive in action, and chronic in course. Urethral stricture, prostatic disease, cystitis from its varied causes, renal

calculus in the ureter or pelvis of the kidney, ureteritis, all act in the above way; and in these several conditions the ascending inflammation through the interstices of the mucous membrane of the ureter reaches the pelvis of the kidney. In this group we have included renal calculi, believing, as we have attempted to show, that these concrements are the products of inflammation, oftentimes ascending in character. But since the pyogenous process associated with them may not have proceeded from below, we must hold that a calculus, irritating and obstructing the pelvis of the kidney, producing congestion and renal retention, invites infection by the blood. It does not, therefore, follow that in calculous suppuration there will be found involvement of the ureter and bladder. In all ascending infections by the ureter the pelvis of the kidney is first invaded. Exceptionally a virulent infection will extend through the papillæ to the uriniferous tubules, and if pus is formed in the substance of the kidney, we have a suppurative pyelonephritis. This may be a hyperacute process; in less virulent infections it may rest as a suppurative pyelitis alone; with retention it becomes a pyonephrosis; later, the cortical substance may participate in the suppuration or atrophy under pressure.

Steven, Newman, Albarran, and Casper have shown that in many cases of inflammation in and around the lower urinary tract the kidney may become infected without the intermediation of ureteritis or pyelitis. In such cases the lymphatic arrangement fully explains how these channels may conduct the infection directly to the kidney-substance. The practical point to be obtained from this knowledge is that many suppurative conditions in the bony pelvis may produce a renal suppuration which the cystoscope or ureteral catheterization would not show.

Renal suppuration may be secondary to contiguous infective processes. Hepatic abscess and burrowing appendicitis are familiar illustrations of this mode of infection. The kidney may derive its infection from the colon; in order that this may occur there must be some affection of the gland which lowers its resistance, either a calculus, hydronephrosis, or contusion or laceration, the result of traumatism. A hydronephrosis is peculiarly liable to become infected from the colon because of the closer relation established by the renal distention. Vinay and Cade

describe a pyelonephritis of pregnancy in which the infection is derived from the intestines, invited to the kidney by its congestion consequent upon pressure of the ureter by the pregnant uterus. A calculus in the cortex of the kidney may induce a plastic inflammation between the capsule and colon; this area permits the migration of germs to the kidney, where, aided by the decomposition of urine, a suppurative process is started.

A pyelitis with accompanying nephritis may follow the irritation produced by the toxines eliminated in the course of specific fevers. Infection which leads to suppuration in the course of these diseases is analogous to that which occurs in septicemia and pyemia, or as a complication of ulcerative endocarditis, and is embolic. The infective embolus is arrested in the renal arteriole, and a metastatic abscess results. Such infections are characterized usually by multiple foci of suppuration, not only in the kidney, but throughout other organs; occasionally a single abscess may form, amenable to surgical relief. The bacilli found in the blood in zymotic diseases, being eliminated in the urine, may cause a suppurative pyelitis, which may become pyonephrotic; Garnier and Lardennois, and Fernet report finding the bacillus typhosus in pure culture in pyonephrosis.

Pathology.— **Suppurative Pyelitis:** The alterations induced by infection of the pelvis of the kidney are influenced largely by the virulence of the infecting agent and the condition of the renal pelvis. Clinical experience teaches that in old cases of prostatic or other obstruction in the lower urinary tract, the impeded renal discharge in some way establishes a tolerance to irritation not borne by perfectly normal structures. All forms of renal suppuration tend to pursue a chronic course. But in pyelitis, whether hematogenous or ascending in origin, the morbid changes may indicate the greatest activity. The mucous membrane loses its lustre, there is rapid shedding of epithelium, ecchymotic spots indicative of the great congestion appear over its surface, and deposits, diphtheritic and purulent in character, line its expanse. The retained urine is turbid from admixture with pus, epithelium, and blood-corpuscles; and coagula of jelly-like consistency, teeming with microörganisms, are discharged through the ureters. In the more chronic cases the mucous membrane is thickened, indurated, in spots may appear granular

or ulcerated, and as a whole partakes of the character of a pyogenic membrane. These changes are especially noted in pyelitis associated with calculi. Between these two extreme grades every shade of varying is found. If the pyelitis has developed from the lower urinary tract, the ureter will evince, in less degree usually, the changes seen in the pelvis. The changes encountered in other parts depend entirely upon the cause. The discharge from a primary or hematogenous pyelitis may initiate a most active infection in the lower urinary tract.

Suppurative Pyelonephritis: This appears in two forms. The acute process involves the entire kidney, while the chronic may invade widely disseminated areas, or be confined even to one pyramid. Both are secondary to pyelitis, the character of the primary inflammation determining the extent and rapidity of the pyelonephritis. When the pelvis is invaded by infection its advance into the calices and uriniferous tubules may be very rapid. The microörganisms are aided in their action by the decomposed urine; they ascend into the tubules, multiply, and with the degenerated epithelium form plugs, which occlude the tubules and undergo coagulative necrosis. These intratubular changes are general to the kidney, and extend from papillæ to cortex. The intertubular substance is the seat of active inflammation, and in advanced cases the whole renal structure is broken down and appears as a granular or purulent mass. If a section of the kidney is made in the early stage of suppurative pyelonephritis, yellowish streaks or cones, base upward, will be seen, indicating the above changes. The cortex of the kidney may show upon its surface minute yellow spots. The capsule is adherent. Here and there throughout the organ may appear more distinctly circumscribed abscess-formations. Venous thromboses exist; the arteries are the last structures to succumb. The microscopical picture, showing the invasion of the tubules by the bacteria, is undoubted evidence of the ascending character of the infection. In more chronic cases only portions of the renal structure may yield to the infection, and localized areas of suppuration, presenting the same features as above described, are seen.

Pyonephrosis: When suppurative pyelitis eventuates in or is attended by ureteral occlusion, partial or complete, the retention

and accumulation of the purulent fluid in the renal pelvis and calices constitutes a pyonephrosis. This is such a frequent complication of calculus-disease that it is a habit of writers to classify it as calculous pyonephrosis, in contradistinction to that due to other causes. It may be transient or permanent, but is necessarily a chronic process. The renal pelvis is found thickened and surrounded by an indurated mass of fatty tissue. Its inner surface presents the same changes met with in chronic pyelitis. The mucous membrane is divested of its epithelium, and may show areas of ulceration; but usually it is smooth, indurated, and of a dull, white color. If leakage occurs, the accumulation may be limited in amount, dilating only the pelvis. In greater distention the calices are effaced, fibrous septa marking their sites, the papillæ are flattened, the glomeruli, if found, are fibroid, the cortex is compressed and forms with the capsule the fibrous wall, irregularly pouched and varying in thickness, and the whole organ is converted into a multilocular cyst containing fluid composed of urine, pus, and tissue-débris, and perhaps calculi. In old cases of pyonephrosis with complete atrophy of renal structure and permanent ureteral obstruction, the watery elements of the contents of the sac may be absorbed, and a butter-sauce-like material remains.

A pyonephrosis may be accompanied by a suppurative pyelonephritis; but usually this complication affects only an area of the remaining secreting structure, and appears as an isolated abscess. When the obstruction precedes the infection, hydronephrosis is the primary condition; infection of the hydronephrotic sac may come from the ureter, but generally it is from the contiguous bowel. The conditions consequent upon an infected hydronephrosis are so different from those that are present as the result of pyelitis eventuating in pyonephrosis that it scarcely seems proper to regard them as one. Their pathological evolution is as different as an abscess of the ovary and a suppurating ovarian cyst. An infected hydronephrosis may be a tumor of enormous dimensions; while pyonephrosis, strictly speaking, may attain to considerable size, it does not approximate to that of infected hydronephrosis.

Renal Abscess: When the kidney becomes infected either through blood or lymphatics, a single abscess may form, or mul-

tiple foci of infection may appear scattered throughout the organ. If the infection follows traumatism, the entire kidney or any part of it may succumb to suppuration. Such renal suppurations are things apart from those we have just considered, and are not initiated by pyelitis. The seat of embolic and lymphatic infections is primarily in the interstitial substance. They encroach upon and destroy the tubular structure, and may acuminate toward the pelvis of the kidney, or beneath the capsule. Multiple abscesses are usually of small size, merely one manifestation of a general pyemic condition. Infection by the lymphatics may be single, yet it, too, appears as a diffuse suppurative interstitial nephritis.

Aside from the causal lesions, suppurative conditions of the kidney beget complications of serious import. If the condition is bilateral, and unfortunately suppurative pyelonephritis often is, anuria is to be feared. Pyonephrosis, though often a unilateral affection, may involve both organs, ultimately leading to atrophy of renal structure and complete arrest of renal secretion. A suppurating kidney is a constant menace to life. A disintegrating thrombus in a venule may prove the medium of remote and fatal infection. A pyonephrosis or a renal abscess acquires adhesions to adjacent structures. Not infrequently rupture occurs, which may be followed by peritonitis, splenic or hepatic abscess, subdiaphragmatic abscess, pleurisy, or pneumonia. Should the escape of pus take place extraperitoneally, a perinephric abscess is formed, which may discharge externally, a possible cure, or as a sequel there may be an annoying renal fistula.

Symptoms.—**History**: The burden and purpose of our rather exhaustive etiology is to show that renal suppuration is a descendant of legitimate parentage; it is not a bastard, and in its infancy occupies a definite home; the closer our acquaintance with its progenitors, the better our comprehension of the nature and behavior of the offspring. No excuse is offered for holding to a classification of renal suppurations; nor is it held that the express pathology occupies definite boundaries; but we maintain that the mode of infection has a material bearing upon the symptomatology as well as upon the course of the disease. He who is most alive to the possibilities of renal suppuration from its

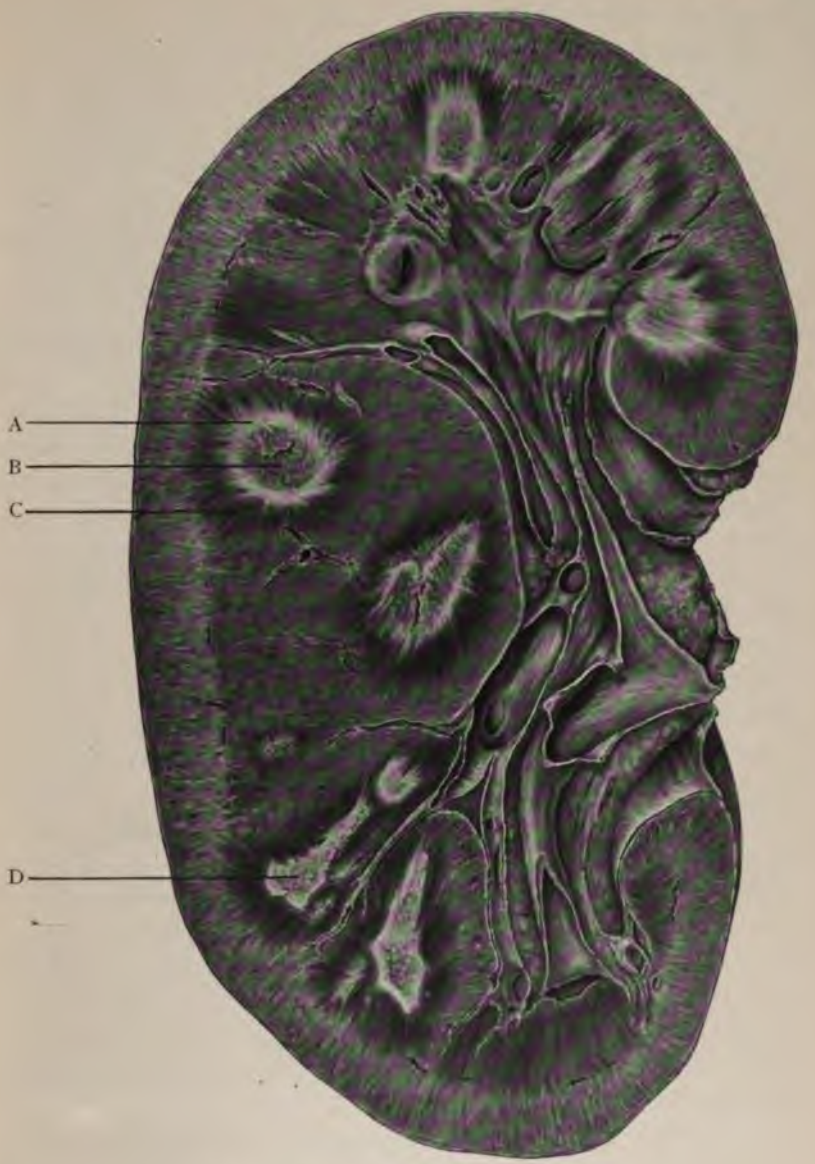
multifarious causes will be the first to recognize its occurrence. Late surgery may save life, but early interference can oftentimes save both kidney and life. For a determinate diagnosis the antecedent conditions equal in importance the special symptoms.

Urinary Phenomena: Renal infection is renal irritation, and within bounds implies increased functional activity. An increased flow of urine is the first, though not always recognizable, symptom of renal infection. When the reaction produced by the microörganisms is slow in its development and of moderate intensity, polyuria may be noticeable for some time. This is conspicuously characteristic of tuberculous infection, and is occasionally observed in the pyogenic processes that we are considering, whether it be an interstitial suppuration or a pyelitis.

Pyelitis excites a frequent desire to micturate, thamuria; the occurrence of this symptom after an apparent subsidence of the irritation in the lower urinary tract, or in those constitutional conditions that occasion pyelitis, is sometimes the first signal of renal involvement. It occurs early as the result of irritation, unaccompanied by foreign elements in the urine, that is, before there is actual suppuration; Osler affirms this and experience teaches it. When plugs of mucus, pus, or calculi enter the ureter its spasm is transmitted to the bladder, and frequent and painful urination follows. Hence it is that frequent micturition is an early as well as a late symptom in all cases of renal suppuration involving the pelvis, so long as the ureter is not occluded.

Frequent micturition does not imply an excessive or even a normal flow of urine. The transient initial polyuria is succeeded by a diminution in the quantity of urine from the kidney involved, but not necessarily a decrease in the total amount voided. In slow infection of one kidney, if the other organ is healthy, it will gradually assume the functions of its crippled mate, and may be capable of discharging the full duties of both. This accommodation can only be extended after a time and very gradually, and therefore there exists for a space oliguria in all those conditions in which there is an impediment to the flow of urine. Pyelitis alone will show it, but usually in an intermittent way. If there is obstruction and consequent pyonephrosis the absence of urine is due not only to the ureteral obstruction, but to atrophy of the renal secreting structure. In suppurative pyelonephritis

PLATE XIII.



KIDNEY. MULTIPLE EMBOLIC ABSCESSES.

A. Interior of abscess with pus in situ.
B. Necrotic periphery between pus cavity and adjacent inflamed renal tissue.
C. Reactionary inflammatory area, surrounding and circumscribing the abscess.
D. An abscess showing something of the fan or radiating contour frequently observed in suppurating infarcts; other abscesses in the specimen show this peculiarity to a moderate degree.

without ureteral obstruction, there is a diminution in or actual suppression of urine from the blocking of the tubules. Interstitial suppurations, either by size or number, destroy the secreting structure of the kidney, and lessen the amount of urine. For clinical purposes it is ordinarily sufficient to measure the amount of urine passed within twenty-four hours, but scientifically we should estimate the separate urines from each kidney.

In bilateral affections, especially in suppurative pyelonephritis, and in double calculus-disease, the oliguria may amount to actual suppression, anuria, a subject fully dealt with under a separate head.

Pain: An isolated abscess in the substance of the kidney may exist without pain. Chronic suppurative pyelitis may cause pain only at intervals. A stable pyonephrosis may be attended by only moderate suffering. Yet pain must be regarded generally as a constant symptom of renal suppurations. It is the most noticeable and guiding symptom in the onset of the disease. It marks the progress of infection by acute exacerbations. Several factors in the morbid process contribute to the production of pain: (1) the tension of congestion, (2) the tension of obstruction, (3) the presence of a concrement, (4) ureteral spasm, (5) the weight and displacement of the kidney, (6) localized peritonitis.

The quality of pain is measured by the several factors that enter into its production. It is described as a backache, a lumbago, a nephralgia, sometimes as an intermittent burning sensation in the side and loin, or as nephritic colic. In one it may be simply a dull ache; this is especially so in chronic affections involving the cortex. In an acute suppurative pyelitis the pain is sharp and cutting at times, with a persistent ache or throbbing. In destructive pyelonephritis the pain may be of moderate intensity, but we must bear in mind that the septic and uremic state of the patient obtunds the sensibilities. Calculous pyonephrosis gives the history of intermittent attacks of nephritic colic. If the obstruction is partial, then the suffering continues paroxysmally; in the intervals there is the discomfort due to the tumor's weight and dragging upon the pedicle, increased by exercise and change of posture, aggravated by constipation and the digestive disturbances so generally present. In large abscesses,

or in pyonephrosis sufficient in size to encroach upon the peritoneum, the pain is sharp and lancinating or burning in character.

The seat of greatest suffering is in the immediate renal region usually, yet it is oftentimes referred and erroneously ascribed to other parts. Its reflection to the bladder and external genitals is common. Its radiation along the genitocrural and sciatic nerves may alone be complained of. It is often felt in the abdomen, and with other phenomena becomes a misleading feature.

Septic Phenomena: Toxic symptoms are never absent, though they are varied in their expression. All acute infections show thermal disturbances, often preceded by a distinct rigor, or chilly sensations. In the onset of pyelitis and pyelonephritis the temperature may mark 104° or 105°, may continue high, but usually recedes somewhat; it is more continuous in pyelonephritis than in other forms of infection.

Pyonephrosis gives the fluctuating chart of sepsis; for long intervals the temperature may be normal, and then a cool stage or a shivering will be followed by fever, these exacerbations usually occurring at night or toward evening; colliquative sweats mark the decline of the temperature. The patient often passes into a hectic state; the skin is dry and yellow, thirst is urgent, and digestive disturbances are marked. The general condition of the patient is one of decline; he is anemic and loses flesh. Secondary suppurations may occur in other organs; endocarditis is a frequent complication. Nervous symptoms may appear, and the headache, delirium, dyspnea, stupor, and coma or convulsions are ascribed to the decomposition and absorption of urinary substances. This *ammoniemia*, as it is called, is probably nothing more than bacterial intoxication. This tableau may be said to mark the closing scene of pyonephrosis. We must bear in mind that this is a chronic affection, and may endure for months, even years, with slight constitutional disturbances, ultimately to take the course above indicated, unless interrupted by some intercurrent affection or complication.

Pyelonephritis, the "surgical kidney," has no such extended existence; succeeding a pyelitis, it pursues a rapid course, and may end in a few days or weeks at most. The high and sometimes intermitting fever, the distinct initial rigor with perhaps

a succession of chilly sensations, the vomiting, the headache, indifference, and lethargy, the typhoid state, a tendency to somnolence, together with a marked oliguria or anuria, point clearly to the seat of the almost certainly fatal affection.

PHYSICAL SIGNS.—**Tenderness**: That all painful suppurative conditions are tender under palpation is a common observation. It is far more significant to note that tenderness may be elicited when spontaneous pain is not complained of. Tenderness may be developed only by fixing the kidney between the palpating hands, and then with the anterior hand making a quick thrust. In the normal kidney this will create a sensation of nausea; if a calculus or abscess is present, the pain will be sharp and lancinating, and often attended by a desire to micturate. Deep point-pressure posteriorly with thumb or finger may cause acute local and referred pains. When a renal affection is extending, as is indicated by the constitutional symptoms, tenderness is more marked. When the tumor advances toward the peritoneum it is habitually tender over its surface.

Muscular Rigidity: An individual bearing a suppurating kidney generally has a stiff back, revealed perhaps more by his carriage than under palpation. An acute infection is generally defended by rigidity of the loin muscles.

Tumor: An infected kidney, owing to its increased weight, is generally displaced downward. Pyelitis and pyelonephritis produce only moderate increase in the size of the organ. Suppurative interstitial nephritis may give rise to considerable swelling. Pyonephrosis is always attended by increase in the kidney's bulk. The tumor may not be easily palpable in moderate distention; or it may form a large swelling, producing distinct bulging of the loin, filling in the costo-iliac space, on the right displacing the liver upward, and on the left the spleen inward and forward. Very large pyonephrotic sacs, which appear as abdominal tumors, are generally instances of infected hydronephroses. If of moderate size, the tumor moves under manipulation, and if adhesions are not acquired, is somewhat influenced by respiration. It is usually irregular or lobulated in outline, may be hard and resisting, or in areas elastic or fluctuating. Auscultatory percussion shows that it is separate from the liver, and will reveal the colon in relation with its inner and inferior borders. Posteriorly

and laterally it is dull under percussion. On account of adhesions it cannot be moved as freely as an uninflamed wandering kidney. Large pyonephrotic sacs are usually very fluctuant, often encroach upon the diaphragm or liver, are covered by the colon, and sometimes by small intestine.

In cases of horseshoe kidney and other abnormalities the physical signs are very misleading.

Cystoscopy: The intravesical picture may reveal the etiology, and in some instances determine the ascending or descending character of the infection. When a cystoscope is in position, especially if its beak touches the mucous membrane near the orifice of the ureter, there may be an absolute interruption to the discharge of urine. This cessation of the flow misled the author upon one occasion into believing that there was a ureteral obstruction. Numerous observers have remarked the inhibiting effect of the cystoscope. The employment of the cystoscope, and through it the catheterism of the ureters, is a practice growing in popularity. Too much cannot be said in commendation of Dr. Howard Kelly's work. Yet in infective processes the greatest care should be used to obtain asepsis of the field through which a sterilized instrument is passed.

Pyuria: In discussing the examination of the urine it is presumed that all precautions are taken to obtain that fluid free from extraneous contamination. The importance of estimating the quantity and quality of urine from the separate kidneys has been emphasized. Unless this has been ascertained by ureteral catheterization, when one ureter is blocked we may be misled by the secretion of the opposite kidney which may be perfectly normal.

It is now proper to consider under what conditions the urine will become contaminated in suppurative affections of the kidney.

Suppurative pyelitis will discharge through the ureter its inflammatory products, perhaps for a time continuously; intermissions may occur, due to a temporary subsidence of the inflammation, or to a transient occlusion of the ureter.

As stated in the pathology, pyonephrosis may be accompanied by complete occlusion of the ureter, or the leakage may be continuous or intermittent. If the obstruction is absolute the vesical urine will be from the other kidney only. If the obstruction

acts intermittently, with a free discharge of pus there is often a reduction in the size of the tumor, and a marked abatement in the symptoms. This constitutes one of the most important and diagnostic signs of pyonephrosis.

In as much as suppurative pyelonephritis is but an extension of a similar process from the renal pelvis, urines from kidneys so affected will contain pus.

An abscess, traumatic or embolic in nature, situated in the substance of the kidney, will not produce purulent alteration in the urine until it opens into the renal pelvis.

Microscopical examination of the urine contaminated by renal suppuration will show pus-corpuscles, abundance of microörganisms, tailed epithelia, and leucocytes. Occasionally in acute, often in calculous conditions blood-corpuscles are seen. Relative to the microscopical diagnostic significance of the renal urine, Prof. Thomas H. Manley makes this rather remarkable statement: "By its revelations alone we may more completely elucidate diagnosis than by all our other resources combined." And he further makes the supporting statement that only the incompetent discredit this method. If its value is based upon an effort to determine the source of the epithelium, it can be asserted upon the highest authority that the discrimination cannot be made between epithelium from the pelvis of the kidney and that from the bladder. Osler, Musser, and Tyson all practically discard the microscope as an aid in determining the source of the pyuria. If casts composed of pus-corpuscles and microörganisms *en masse* are found, it is strongly presumptive of pyelonephritis.

Chemical Examination of Urine: The usual infecting germ is the bacillus coli communis, and with it we have acid urine. The statement is too generally made that urine containing pus from the kidney is always acid. Calculous pyuria is not infrequently alkaline in reaction. While pus may be present without showing albumin, usually urine containing pus is albuminous.

DIFFERENTIAL DIAGNOSIS.—In urinary disturbances with pyuria the paramount question is to determine the source of the pus. In the body of the article we have indicated the means of doing this, by obtaining the separate urines from the ureters, thus elim-

inating inflammations of the lower urinary tract, or showing their extension to the ureter and kidney.

When the complexus of symptoms indicates renal suppuration, it is a very material question to decide the nature of the pathologic process. Is it an ordinary case of ascending infection, or is it infection incident to calculus, or is it a specific infection due to the bacillus tuberculosis? Upon our ability to decide these three points rests the intelligent management of the case. In ordinary infections arising from obstructive conditions in the lower urinary tract, the history of the case and its clinical course indicate usually the primary seat of the disease. If uretheral or vesical instrumentation has been frequently employed, it is safe to assume that ample opportunity has been offered for colon-bacillus infection; and we may further apprehend a simultaneous infection in both kidneys. In calculous conditions the chronicity, the attacks of nephritic colic, the negative examination of urethra, prostate, and bladder, a cystoscopic and urinary examination localizing the affection in one kidney, and the confirmation of the Röntgen ray are usually sufficient to make the diagnosis. Renal tuberculosis has a rather definite clinical behavior. The detection of the tubercle-bacilli in the renal urine is conclusive; but unfortunately this evidence is frequently wanting.

Hydronephrosis: The physical features of the retention-tumors are very similar. The septic symptoms, and possibly pyuria, distinguish pyonephrosis from hydronephrosis.

Typhoid Fever: Suppurative pyelonephritis reduces the patient to a state which may easily be mistaken for enteric fever. The mode of onset with its attendant conditions, the urinary haps local signs, will disclose the true nature of the affection. In doubtful cases the Widal test should be made.

PROGNOSIS.—Bilateral affections are always serious. Acute infections attended by high fever and diminished urine are of gravest import. Calculous pyonephrosis, the other kidney being healthy, is the most amenable suppurative condition. Suppurative pyelitis from all causes may temporarily improve; a most guarded prognosis should be given concerning its ultimate course. Embolic abscess may be single, and may involve only

one kidney, in which case it can be successfully treated. Pyemic suppurations are generally regarded as hopeless conditions; Lilienthal reports recovery after a double nephrotomy for multiple abscess. In obstructive conditions associated with renal suppuration the prognosis depends upon our ability to remove the cause.

SURGICAL INDICATIONS.—The surgeon is called upon to interfere in infective renal conditions to prevent death from uremia and acute sepsis; in more chronic states, especially those accompanied by retention, to relieve suffering, and to rescue the secreting structure of the kidney from atrophy or advancing suppuration; and finally, when the organ is destroyed, to rid the body of the cause of suffering and source of poisoning.

Suppurative pyelitis does not always require surgical interference. When it originates in the course of zymotic diseases, it yields to constitutional measures. The topical treatment of pyelitis through the ureteral catheter does not appeal to the practical surgeon.

In acute infective pyelonephritis *nephrotomy*, single or double, as is demanded, is a simple procedure which may, by relieving the tension and affording drainage, save life under desperate circumstances.

In chronic conditions the first indication is to determine the efficiency of the other kidney, a knowledge that will safeguard us from possibility of a humiliating disaster. It is no easy matter to determine whether nephrotomy or nephrectomy should be done. If both kidneys are involved, *nephrotomy* only should be thought of. If there is no ureteral or renal obstruction, *nephrotomy* should be chosen in all cases in which there is evidence that the kidney still possesses secreting function. If the obstructing cause can be removed, *nephrotomy* as a primary procedure, at least, should be tried. Clear conducting channels justify tentative measures. When the kidney has been destroyed by atrophy or suppuration, *nephrectomy* is called for. When ureteral obstruction is insurmountable, *nephrectomy* may be the desired primary procedure; it will ultimately become necessary.

Abscess, traumatic or embolic, requires *nephrotomy* only, unless the entire organ has been converted into a pus-cavity, and then *nephrectomy* alone will suffice.

Calculous conditions should be subjected to incision, removal of the concrement, and drainage; or if the stone is in the ureter, to *ureterotomy,* with *nephrotomy* if demanded. In those cases where it is apparent that the kidney is destroyed by calculus-disease, primary *nephrectomy* should be done, the efficiency of the other kidney and the patient's general condition warranting it. In renal suppurations consequent upon ureteral obstruction by calculus or by stricture, with total destruction of kidney-structure, *nephro-ureterectomy* is indicated.

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TUBERCULOSIS OF THE KIDNEY.

DEFINITION.—A primary or secondary infective disease due to the bacillus tuberculosis, pathologically active and symptomatically localized in the kidney.

SYNONYMS.—Phthisis renum, nephrophthisis, scrofulous kidney.

FREQUENCY.—Morris shows by an examination of the records of the Middlesex Hospital that of 2,610 necropsies primary renal tuberculosis was met with in 15 cases. Brown, from Thacher's work at the Presbyterian Hospital, New York, notes that of 567 autopsies 68 bodies showed tubercular lesions; 23 of these had tuberculous kidneys. It is shown to be relatively more frequent in the kidneys than in the liver or spleen, yet the analysis does not seem to have determined the number of cases that were primarily renal. Perhaps the most practical conclusion as to the frequency of renal tuberculosis may be drawn from the statistics of the operations undertaken for its relief. Facklam was able to gather together 108 cases reported up to 1893. Bangs, excluding all of Facklam's cases, has collected, during the decade 1888 to 1898, 135 cases subjected to operation for renal tuberculosis. With the attainment of greater precision in diagnosis, the recognition of localized renal tuberculosis and its surgical treatment are of yearly increasing frequency.

ETIOLOGY.—**Age**: The disease is more common between the ages of twenty and forty years.

Sex: Israel states that female patients contribute 60 per cent. of all his operative cases of tuberculosis of the kidneys, and that 75 per cent. of all cases of primary renal tuberculosis are in women. These figures are in close accord with those of Facklam and of Bangs. Of 28 operations for renal tuberculosis performed by Mr. Morris, 8 were on males, 20 on females. It therefore appears statistically that it is almost three times as common in females as in males. The comparative ease with which the female bladder and ureters may be explored no doubt accounts for its greater frequency of recognition in that sex.

Miliary renal tuberculosis occurs as an epiphenomenon of general miliary tuberculosis, a condition not amenable to surgical treatment and with which we are not concerned. Truly speaking, primary renal tuberculosis can only occur in one way, and that is by infection through the blood. The bacillus is taken up from the mucous or cutaneous surfaces by the lymphatics, advanced to the veins, then to the arteries, and by one of the renals is brought to the kidney (Brown). Under local predisposing conditions of congestion from previous disease or displacement, or irritation from calculus, infection takes place, developing a purely primary tubercular granulation. An inherited susceptibility contributes to the possibilities of tuberculous reaction. Renal tuberculosis from infective emboli must be classed as secondary, though the supplying focus may be latent. In the hematogenous varieties the primary site of the disease is in the parenchyma of the organ. Both of the above forms of renal tuberculosis are described as descending infections, for with the eruption and discharge of the tubercular nodules into the pelvis of the kidney, the ureters and lower urinary tract develop the disease. In other instances the primary tubercular lesion is located in the genitals or bladder, and infection ascending in character, either by the mucous membrane of the ureters or through the medium of the lymphatics, reaches the kidney and there excites, usually in the pelvis, a tuberculous process, which may involve the whole secreting structure of the kidney. Renal tuberculosis may very exceptionally be due to infection from tuberculous disease in contiguous organs.

Pathology.—When the bacillus is conducted to the kidney by the blood, it is presented by the terminal arteries to the glomeruli, there to be returned to the circulation by the veins or else eliminated; manifestly, therefore, that part of the kidney containing the glomeruli, namely, the cortical substance, is the most common seat of hematogenous tuberculous infection. The lodgment of the bacillus excites a reaction resulting in local changes due directly to the bacillus, and remote lesions the result of the absorption of its toxines. Renal tuberculosis does not differ materially from a similar process in other organs. There is a cluster of giant-cells and granular *débris* in which a few bacilli may be found, surrounded by large cells with big nuclei, often called

KIDNEY, MASSIVE TUBERCULOSIS.

The entire secreting substance has been replaced by caseous detritus. The pelvis shows miliary granulations and the ureter is greatly thickened. The outlines of the pyramids are indistinctly preserved.

epithelioid cells, the whole encased in lymphoid elements. This lesion, at first microscopic, becomes by a coalescence of several, a yellowish-white or gray tubercle visible to the eye. Metchnikoff and Borrel maintain that a tubercular granulation is an aggregation of phagocytes, a lymphatic formation and not a proliferation of fixed connective-tissue cells.

In the kidney the process is primarily interstitial, and the renal epithelium plays no active part. The growth of the single tubercle, or the coalescence of several single lesions leads by coagulative necrosis to the formation of a cheesy nodule. There may be one or several foci of infection and degeneration originating in the cortical substance, which usually assume a pyriform shape, invade the medulla, and approaching the cavity of the renal pelvis, rupture into it. Exceptionally, circumscribed collections evolute toward the surface of the kidney, elevating its capsule, and sometimes bursting through and giving rise to perinephric suppuration. When the entire kidney succumbs to the caseous degeneration, its secreting structure entirely destroyed, its capsule enclosing only a putty-like mass of material, it is described as massive tuberculosis. These aggregated tubercles, breaking down, form cavities, the so-called tubercular cysts, with a limiting wall of embryonal tissue and numerous blood-vessels. The contents is caseous matter of a yellowish-gray color, or mixed with blood, of decided urinous odor, and in very chronic cases with incrustations of phosphate of lime. Tubercle-bacilli may not be found in the contents of these cavities but always in the walls. If mixed infection has occurred, other pyogenic organisms are found. The calices and pelvis of the kidney become secondarily involved, either by eruption of nodules or by contiguous infection. With the advance of the tuberculous process and the discharge of the caseous masses into the pelvis of the kidney, occlusion of the ureter leads to the development of a hydro- or pyonephrosis; however, a cystic degeneration of the kidney has been shown to occur without ureteral occlusion (Klippel).

With the discharge of the tubercular matter into the pelvis of the kidney, we have the phenomena of descending infection; there is a pyelitis in which the pelvis may show tubercular granulations or be deeply ulcerated; the ureter is thickened, ulcerated,

or strictured, showing here and there irregular dilatations, but usually contracted into a rigid tube. In severe infections tubercles appear in the mucous membrane of the bladder about the ureteral orifices, and in the trigonum and fundus of the viscus. These, breaking down, form the so-called lenticular ulcers, characterized by a flat base covered with caseous matter, and sharp, ragged borders within which little miliary nodules are situated. It is only in advanced cases of descending tuberculosis that these gross changes in the bladder are observed. In the early state the mucous membrane of the bladder may appear healthy everywhere except at the mouth of the ureter supplying the infection, where it may only show increased turgescence; or clearly defined areas of inflamed mucous membrane may be seen between it and the slightly hyperemic trigonum, leaving interposed tissue unchanged in appearance. Further infection of the prostate, seminal vesicles, urethra, and testicles occurs. Brown records a beautiful illustration of primary renal tuberculosis of one kidney which ultimately, by ascending infection, involved the other ureter and kidney. Newman refers to a similar observation reported by Coats.

Ascending renal tuberculosis or infection by invasion along the lymphatics of the kidney from foci of infection in the lower urinary tract is regarded by Newman as by no means uncommon. It is universally stated that infection of the kidney takes place frequently from the bladder along the ureter. Baumgarten, after much experimental work, asserts that this form of infection never occurs. It was the consensus of opinion of the genito-urinary surgeons at the 13th International Medical Congress, at Paris, that renal tuberculosis is often primary, and that bladder-tuberculosis is regularly secondary to some prior localization of the infection.

The etiology of genito-urinary tuberculosis is, as yet, somewhat obscure, and its pathology exceedingly complex. It is more than probable that further investigation will not only confirm the existence of a primary renal tuberculosis, but will further show that tuberculous disease of the lower genito-urinary tract is secondary to renal tuberculosis. In proof of this we might cite the undisputed clinical observation of the obstinacy and resistance to treatment of tubercular ulcers of the bladder,

and the rapidity with which the self-same ulcers heal spontaneously after nephrectomy for tuberculous kidney. In ascending tuberculous infection the pelvis of the kidney is first invaded, and the patient not infrequently succumbs before the disease has advanced to the cortical structure. This pyelitic tuberculosis, so called by König, is regarded as indisputable evidence of the ascending form of infection, in contradistinction to the cortical tuberculosis of hematogenous source.

Tuberculous infection does not always result in necrosis and degeneration of tissue. Widal and Benzançon have recently described a form of tubercular sclerosis due to mild infection. Other changes in the kidney, such as nephritis and amyloid degeneration, are attributable to the irritation of the epithelial cells by the toxines. The tubercular inflammation results in thickening of the capsules, and often in acquired adhesions. The gland is usually enlarged, though a palpable enlargement is said by Newman to occur only in about one-fifth the cases; Israel regards it one of the most positive signs of tubercular inflammation. Enlargement is rarely detected in the early stage; it usually results from ureteral occlusion and accumulation in the pelvis.

Symptoms.—The clinical recognition of renal tuberculosis is beset with difficulties. Happily, they are not insurmountable when one is prepared to conduct the inquiry in a scientific way; but unfortunately, few surgeons are familiar with the details of the technic essential for diagnosis in the early stage of the disease, that period which offers the highest reward to the surgeon's art. It must be emphasized that miliary tuberculosis involving the kidney is not a surgical affection. We are occupied only with the consideration of what is known as chronic renal tuberculosis, "scrofulous kidney," and chiefly with the primary renal localization; secondary or ascending infections are only exceptionally amenable to radical surgical measures. It is, therefore, apparent that, when confronted with symptoms indicative of kidney-disturbance of possible tuberculous origin, we must first survey the entire man to discover if there is an active focus of disease which may be supplying infection for general dissemination. If such be found, but small hope would attend any surgical measure undertaken for the relief of any special renal pathology. With

the exclusion of general tuberculosis, the problem is not solved. It yet remains to determine whether the disease involves primarily the kidney or is an ascending infection, and here the superficial examiner is misled by assuming that functional disturbance always indicates the seat of pathological change, that is, incriminating the bladder on the evidence of frequent urination; or else he accepts consecutive or complicating lesions as sufficient explanation of the symptoms. In the pathology we have shown how frequently and how certainly descending infection will involve the bladder and genitals, and how it may even ascend to the other kidney. It cannot be gainsaid but that the more localized a tuberculous infection is, the greater the surgical opportunities. It is, therefore, imperatively demanded that we recognize in its early stage primary renal tuberculosis.

History: We must take into account the family anamnesis. An inherited dyscrasia or predisposition appears frequently in the clinical history of chronic renal tuberculosis, and due consideration must be accorded this fact. Renal irritation or congestion creates a *locus minoris resistentiæ,* and as these conditions attend a preternaturally movable kidney, and furthermore, as tuberculosis and movable kidney are both more common in women, this abnormality may, in some measure, explain the renal localization. Insidious in its onset and chronic in its course, renal tuberculosis may exist for some months in those who are to all appearances vigorous and robust. Indeed, active local symptoms may be presented by one of apparently unimpaired health. Slowly, but surely and progressively emaciation makes itself evident; it is not noticeable during the early stage of the disease, but it becomes conspicuous with the appearance of pyuria and with the advance of the disease to the ureters and bladder. Brown attaches the greatest importance to the pallor as an early symptom in these patients; exceptionally it may be due to the constant hematuria, but as it is present also without hematuria, it must be explained as a cachexia, the result of absorption of the toxines.

Polyuria: With the establishment of a tubercular focus in the kidney the gland is stimulated and there is excessive secretion of urine. Frequent micturition and a copious discharge of a clear limpid urine, especially at night, is one of the earliest, often

disregarded symptoms of renal tuberculosis. This apparently causeless and painless abundant flow of urine, with diabetes and hysteria eliminated, should suggest renal tuberculosis in its early stage. It is more significant than thamuria, frequent urination, a symptom of inflammation of any part of the urinary tract. When a greater portion of the secreting structure of the kidney is involved in the degenerating process, and the nodules break down and open into the pelvis, there will still be frequency of urination, but polyuria gives way to oliguria and dysuria, and, too, there may be marked alteration in the character of the urine.

Hematuria: Pousson believes that tuberculous kidney, like pulmonary phthisis, passes through a stage of congestion before it reaches suppuration. As a symptom of this congestive stage we may find blood in the urine. Hematuria in association with pyuria is rather a constant symptom of the advanced stage of renal tuberculosis, indicating a solution of continuity in communication with the pelvis. Profuse hemorrhages may occur in this stage. Spontaneous hematuria, that is, blood in the urine not provoked by exercise or manipulation, may appear from a tuberculous kidney months before the development of any other symptom and without the show of pyuria. Bearing this in mind, the detection of even a microscopical amount of blood intimately mixed with urine demands further urinary examination.

Pain: Pain of a dull, aching character, felt in the lumbar region, of limited radiation, and described as a lumbago or backache, affecting the carriage of the patient, producing an awkward gait, even lameness, may be the initial symptom of parenchymatous renal tuberculosis. It may not be preceded or accompanied by hematuria. Such a pain, enduring for years, in Morris's case for sixteen years, and in one of my own for five years, may be attributable not to the existence of tuberculous disease during all this time, but to some one of the predisposing conditions upon which tuberculosis is engrafted later. And we must not forget the observation of Habershon, that aortic valvular disease may produce renal pain. A caseous nodule slowly invading the parenchyma and distending the capsule will produce the dull, heavy, dragging pain of tension. Should hemorrhage occur in this stage, the passage of clots along the ureter would excite paroxysmal and characteristically referred pains. With the eruption of

the caseous mass into the pelvis of the kidney, pain becomes severe, intermittent, or paroxysmal, radiates to the genitals, sacrum, and thighs, and often to the other kidney; it is rarely referred to the epigastrium or thorax. The sharp, nephritic, colic-like pains are attributable to the passage of masses of caseous matter through the ureter, and to the partial occlusion of that duct by the foreign substance or by inflammatory thickening. There is more or less suppression of urine during the intense paroxysms of pain; with a free flow of pus and urine, the suffering subsides. In the intervals between these exacerbations of pain the patient is not entirely comfortable. Should hydro- or pyonephrosis develop, there is persistent pain with local tenderness. In the advanced stage vesical irritation, frequent micturition and tenesmus are the distressing characteristics of the pain; and often these symptoms are present when the surgeon is first consulted. To recognize tuberculosis at that time is comparatively an easy task, but unfortunately we have permitted the development of complications and conditions which may preclude recovery.

Hectic Phenomena: We have previously alluded to the pallor and emaciation, attributable to the absorption of toxines. In the early stage of primary renal tuberculosis, if the patient is carefully watched, a slight vespertine pyrexia will be recorded. Sweating may occur. These septic symptoms, however, become especially prominent with the discharge into the pelvis of the kidney, when there is liability to mixed infection. If occlusion of the ureter should occur, and there is accumulating pyonephrosis, high temperature, chills, and sweats denote the septic state. The extremities may become edematous. The pain and frequent urination contribute much to the exhaustion of the patient.

Digestive Disturbances: These are not as marked in the early stage of renal tuberculosis as in other forms of kidney-disease. Loss of appetite, constipation or diarrhea are sometimes present. In the paroxysms of pain and in retention, vomiting with great nausea often appears.

Tumor: I have observed increase in the bulk of the kidney in the early stage of cortical tuberculosis before the appearance of pyuria. Palpation will disclose tenderness, but rarely enlargement in this stage. When there is perceptible enlargement it is

regarded by Newman and by Israel as indicating pyelitic involvement with retention. A tumor is, therefore, only very exceptionally an early sign, frequently met with, however, in the latter stages, due to ureteral occlusion.

Urine: In the early stage of primary renal tuberculosis there is abundant urine, clear, of low specific gravity, and of slightly acid reaction; unless there is an associate nephritis, albumin and tube-casts may be wanting. When there is a communication between the tubercular area and the pelvis of the kidney, the free discharge of cheesy matter, detritus, and blood renders the urine turbid and shows a grayish, chalk-like, sticky deposit; albumin is usually present in such urine. "When we can clearly differentiate between globulins and true albumins," says Brown, "the symptom albuminuria in tuberculous nephritis will be settled." There is no sugar, usually a decrease in urea, and an abundance of phosphates. Microscopically, leucocytes, fat-globules, red corpuscles, and shrunken renal epithelia are found. Though the bacilli tuberculosis are regarded as non-pyogenic organisms, yet in all open tubercular lesions in the kidney or elsewhere there is a discharge of pus; and the pus in the urine from a tuberculous kidney has three characteristics, according to Cumston: it is spontaneous, constant and lasting; it may vary in quantity, but is usually an indication of the progress of the tuberculous disease. In old cases bacteriological examination will usually show bacteria, not infrequently the colon-bacillus. But unless the bladder has been infected by instrumentation or otherwise, the pus is often sterile to ordinary reagents. The finding of the bacillus Kochii in urine containing pus is conclusively diagnostic of a tuberculous process in some part of the urinary tract. It has been claimed that tubercle-bacilli have been found in urine otherwise normal, which are derived from a tubercular focus in bone, and which have filtered through without infecting the kidney. The pyuria may be constant, abundant and increasing in quantity, bacilli may be found, the urine may be acid, all of which only shows that there is a tubercular focus somewhere in communication with the urinary tract, yet it is not conclusive at all that it is in the kidney. To obtain this indisputable proof, other means must be resorted to.

Genital Examination: All patients presenting pyuria must be subjected to thorough examinations. In women the vulva, vagina, uterus, tubes, and ovaries must be investigated. In males the testicles, epididymis, seminal vesicles, and prostate must be examined.

Cystoscopy: The inspection of the internal surface of the bladder, rendered possible by the use of the cystoscope, opens the way for a determinate diagnosis. By the view thus obtained we eliminate the ordinary diseases of the bladder which may give rise to confusing symptoms, and oftentimes can declare from the picture presented the tuberculous nature, and reasonably infer the seat of the disease. American observers, Brown, Ramsay, Kelly, and Meyer especially, have become very familiar with the cystoscopic picture of renal tuberculosis. In some cases the opening of the ureter is surrounded simply by a zone of turgescence; in more advanced cases there are circumscribed inflamed spots with healthy areas between, looking like footprints in freshly fallen snow (Meyer). In still more advanced cases the typical lenticular ulcers are seen. The ureteral orifice may be dilated or contracted; in Noble's case, closely studied, it was widely patulous. From the infected ureter jets of turbid urine escape; through the cystoscope, without collection, we may sometimes distinguish its purulent character by contrasting it with the normal urine from the healthy ureter. The normal discharge of clear urine from one ureter is presumptive evidence of the efficiency of that kidney. Renal hematuria may be diagnosed by the cystoscope when other means of determining its source are insufficient. In advanced renal tuberculosis there is not infrequently occlusion of the ureter, a condition that can be determined by cystoscopy alone and with great accuracy if we have taken the precaution to administer methylin blue an hour or so before making the bladder examination. If the ureter is pervious jets of blue urine are discharged from its vesical orifice. We are chiefly indebted to Howard Kelly for the perfection of the technic of cystoscopy and for the additional method of diagnosis, ureteral catheterization.

Ureteral Catheterization: Valuable as all the symptoms and signs of renal tuberculosis above enumerated are, and they are often sufficient to warrant a diagnosis, yet a positive and precise

diagnosis of renal tuberculosis must rest upon a chemical, microscopical, and experimental study of the urine obtained from the separate kidneys. The dangers that attend catheterism of the ureters are strongly emphasized by König, Fenwick, McMurtry, Morris, Musser, and Mayo. It is an operation not without a mortality of its own; even Israel has had a case of severe infection from it. If undertaken through an infected field, it is scarcely possible to obtain safe asepsis, and hence, if the diagnosis of tuberculosis of the kidney can be positively made without catheterism, it should be done. The observance of the technic so widely taught by Kelly, in a measure, safeguards against danger. Through the ureteral catheter we may aspirate even the pelvis of the kidney; and the isolated urine thus obtained contains the key to the diagnosis. If Koch's bacilli are found upon microscopical examination the verdict of renal tuberculosis may be rendered. In chronic, profusely suppurating cases the bacilli are frequently not found; there yet remains a test which should be employed. If this urine is injected into the pleural or peritoneal cavities of the guinea-pig, a very susceptible animal, if the bacilli are present, the pig will sicken and die of tuberculosis. If time and opportunity permit of this experimental test, it eliminates the possible error of mistaking the smegma bacillus for the Koch bacillus, and it is a scientific confirmation of the diagnosis. From the examination of the separate urines from each kidney we not only determine positively or negatively the existence of tuberculosis, but furthermore whether one or both glands are involved, and lastly we may estimate the secreting capacity of the unaffected kidney, upon which depends the justifiability and nature of the operative procedure.

DIFFERENTIAL DIAGNOSIS.—The diagnosis of renal tuberculosis must rest upon the detection of the bacillus tuberculosis in the centrifugalized urine, or upon the proof furnished by experimental inoculation. As the former is uncertain, and the latter necessitates delay, in the absence of these conclusive tests we suspend a precise diagnosis and act upon the surgical indications. Of greatest importance is it to determine that the supposed renal tuberculosis is the only active focus. Exclusion by careful general examination must be insisted upon.

Renal tumors and **hemorrhagic pyelitis** in the early stage are

confusing. The age of the patient, the development of a palpable tumor, its rapid growth, will exclude the former. The latter does not show the general hectic symptoms so important and conspicuous in tuberculosis.

Suppurative conditions due to **calculus** are characterized by severe pain; this symptom is a relative one, and cannot be relied upon for differentiation. The X-ray is a fairly positive test. The history and the course of the disease are more to be relied upon.

PROGNOSIS.—The tendency of primary renal tuberculosis is to destroy the secreting structure of the kidney, impair the permeability of the ureter, infect the lower urinary and genital tracts; to ascend and involve secondarily the other kidney; to supply bacilli for general dissemination and infection; to produce septicemia or uremia. With these possible dangers presented, renal tuberculosis must be regarded as a very serious disease. That it may spontaneously heal, post mortem examinations occasionally show; Tuffier, and Newman, both active advocates for surgical intervention, admit this possible favorable termination. Madelung describes cases of healed nodules. All internists strongly contend for the benefits of hygiene and climate. Bangs, who has most thoroughly considered the subject of prognosis, says: "I think the opinion, based upon such statistics as I have been able to get, is warranted that operation affords better remote results than hygiene." To his mind there are evidently two sides to the question. One needs only to have the clinical care of a case of renal tuberculosis, to watch it to its fatal termination to be convinced that, if surgery offers any possibilities, it should be eagerly grasped. The extremely chronic course, the unimportant and frequently unobserved early symptoms permit the disease to reach a stage that interdicts surgery. The entire question of prognosis, medical or surgical, rests upon an early recognition of the disease. At this period in the growth of the renal bacillosis, hygiene and climate may accomplish something in a very small, indefinite number of cases. Surgery at this time will cure permanently 55.5 per cent., prolong life in 30 per cent., and perhaps abbreviate it but a few months in the remaining. In advanced cases we must rely upon surgery alone for temporary relief. The conclusion is apparent, therefore,

that renal tuberculosis is an eminently surgical disease within certain restrictions.

CONTRAINDICATIONS TO OPERATION.—Merely for the purpose of emphasizing that we have here treated only of chronic or localized renal tuberculosis, it is again stated that general tuberculosis with infection of the kidney is not a surgical disease. Operation is, therefore, contraindicated in all cases of renal tuberculosis in which there is an active focus of tubercular inflammation in other organs; from this broad statement we must reserve the bladder as a notable exception. When tuberculosis of the bladder occurs secondarily to a renal infection, abundant clinical notes show that the secondary lesions heal after the removal of the primarily affected kidney. Weir had an absolute recovery after removal of one-half the bladder, nephrotomy, and later nephrectomy for tuberculosis. It is not unreasonable to predict that infection of the prostate and seminal vesicles will kindly heal after the removal of the focus of supply, or, at least, be more amenable to treatment.

SURGICAL INDICATIONS.—The surgical effort for the relief of renal tuberculosis involves one of four procedures: *nephrotomy, partial nephrectomy, nephrectomy, nephro-ureterectomy.*

Nephrotomy, usually classed as a curative measure, can scarcely be regarded as such. Ramsay can find only 4 cases in which a cure lasting any length of time has been reported. To these I add one of my own of undoubted character; the patient is in excellent health five years after the operation. The objections to nephrotomy are the persistent fistula, the danger of mixed infection, the failure to drain all the infected portions of the kidney; and its good effects are only temporary. Yet as a palliative measure nephrotomy is an exceedingly useful procedure under some conditions. A patient suffering with a large renal abscess may be in such an exhausted state as to preclude radical operation. Nephrotomy with drainage is a life-saving measure, an operation of emergency, and should be undertaken only as such. If the patient recovers sufficient strength, and is not cured, nephrectomy may be done later. My experience, though limited, justifies me in saying that a nephrotomy greatly enhances the difficulties of a secondary nephrectomy. If both kidneys are diseased, nephrotomy is sometimes warranted for

the relief of local symptoms. When our diagnostic measures have determined pronounced tubercular localization in one kidney, the condition of the other being in doubt, it has been suggested that exploratory nephrotomy is justifiable (Küster).

Partial Nephrectomy: Kümmell has shown experimentally and clinically that partial nephrectomy is possible, and it would appear that his beautiful technic would find application in renal tuberculosis. Morris reports a recovery following resection. At operation it is impossible to define the limit of infection. Partial nephrectomy is, therefore, an uncertain and dangerous procedure, and not to be recommended.

Nephrectomy: In all cases of localized renal tuberculosis in which the patient's general condition will permit, and in which the other kidney shows secreting sufficiency, the operation of removal of the kidney, nephrectomy, is the indicated procedure. Tuffier regards it as the only operation to be considered. Hematuria, pain, and infection are his accepted indications for it; he has performed it four times when pain was the only symptom. His experience in the last ten years not only shows the value of surgical intervention, but has convinced him that renal tuberculosis does occur as a primary disease. Ramsay's statistics, which are recorded in 1900, the latest published, of 191 cases of nephrectomy for tuberculosis, show 106 complete cures, counting as cures those cases which, two months after operation, show no symptoms of bladder involvement, or of tuberculosis in other portions of the body. The reported times of observation range from two months to twelve years. Of the remaining 85 patients, 31 improved; 37 died within one month after operation; and 17 died at periods later than one month. Considering all patients that died within one month as operative mortalities, which is manifestly unjust, we find that the mortality is 19.04 per cent. Among the causes of death we find that uremia was responsible in 9 cases. Our improved methods of testing the capacity of the other kidney will greatly lessen this danger and will improve the recovery-rate. This investigation should always be made with the greatest care and thoroughness, as described under Renal Calculus. Simon, in summarizing the results of Czerny's experience with 35 cases of tuberculous kidneys, says that that surgeon has had only 3 operative deaths; 48.5 per cent. of his patients are still living, and

37 per cent. are perfectly well. The lumbar route is almost universally chosen by surgeons in preference to the transperitoneal.

Nephro-ureterectomy: In renal tuberculosis the ureter is so frequently involved in the diseased process that it often gives rise to fistula or abscess when left. Surgeons are now inclined to make the operation as complete as possible, hence it is desirable to remove a portion of or the entire ureter. Of complete nephro-ureterectomy Bovée collects 13 cases with one death. Kelly's work in this line has been exceptionally excellent. Dr. George H. Noble, of Atlanta, reports the removal of the kidney and ureter in one piece by the extra-peritoneo-vaginal method, a unique operation, showing a nicety of technical skill that does him honor; his description of the technic is concisely and clearly detailed. When feasible the entire ureter should be removed, excising the portion of the bladder through which it passed, or inverting the stump into the bladder. Nephro-ureterectomy is a much more serious undertaking than simple nephrectomy; it greatly prolongs the operation and increases the danger of shock.

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FIG. 1.

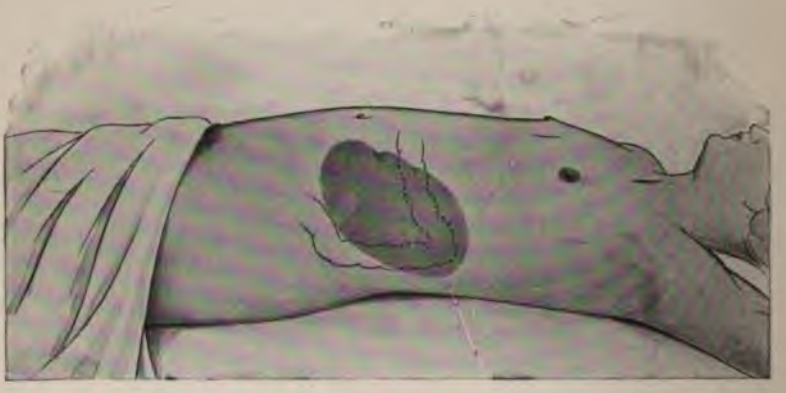


FIG. 2.

USUAL POSITIONS OF COLON IN RENAL AND SPLENIC ENLARGEMENTS.

FIG. 1, Renal tumor showing usual displacement of colon.
FIG. 2, Splenic tumor overlying colon.

TUMORS OF THE KIDNEY.

In the consideration of renal tumors we purposely discard all classifications, awaiting the more mature conclusions of pathologists, and treat the subject more from a clinical standpoint, briefly reviewing the etiology and pathology of such neoplasms only as are of surgical importance. We cannot always differentiate by symptoms and signs the several forms of renal growths, nor can we determine their benignity or malignancy; but this is not of practical importance, for all recognizable renal tumors are malignant in the sense that they tend to destroy the organ involved, and encroach upon adjacent parts, materially imperiling life.

In the following arrangement, after an introduction into the nature of renal tumors, their general symptomatology, with such individual differentiation as is possible, is given.

I. Malignant Tumors.

Definition.—Malignant neoplasms of surgical importance occurring in the kidney are either sarcomata or carcinomata. A *sarcoma* is a connective tissue tumor of predominating cellular structure. A *carcinoma* is an atypical epithelial neoplasm, its cellular elements interspersed in a network of connective tissue stroma.

Frequency.—Among 1,400 cases examined at Manchester Royal Infirmary, Kelynack found 6 cases of primary renal growth. Of 4,505 pathological records, in only 9 were primary renal tumors found. Sarcoma is considered much more frequent than carcinoma.

Etiology.—**Age:** Sarcoma, although not peculiar to, is far more common in infancy and childhood. 80 per cent. of the cases occur under five years of age. Occasional cases occur in those of advanced age.

Carcinoma is met with usually in elderly persons. Newman found the average age in 23 cases to be forty-eight years.

Sex: Birsch-Hirschfeld claims that sarcoma is somewhat more common in girls. Of 130 cases analyzed by Walker, 55 were in

boys, and 51 in girls; of 24 sex not given. Kelynack considers females somewhat more predisposed to sarcoma.

All monographers very studiously avoid the subject of the etiology of malignant tumors of the kidney. Analyses of cases show that neither traumatism, irritation, nor heredity have any bearing upon their production. Cohnheim's theory of embryonal inclusion seems to be clinically substantiated in a number of cases; beyond this we cannot go. Calculi are found in not a few instances associated with carcinoma; their relationship does not parallel in frequency the association of gall-stone with cancer of the biliary channels.

PATHOLOGY.—Malignant disease of the kidney may be either primary or secondary. As compared with other organs, secondary growths in the kidney are infrequent. When both organs are simultaneously affected, the tumor in one or both is probably metastatic.

1. **Sarcoma**: Sarcomatous tumors may originate in any portion of the kidney; they most frequently develop either in the cortex or in the medullary portion of the gland, from the perivascular or intertubular connective tissue. In other instances the growth appears to spring from the capsule, and growing away from, or into the substance of the organ. They may also spring from the tissues of the hilum. The relation of the kidney to the growth cannot be ascertained ordinarily in cases that have progressed to large tumor-formation. A small tumor may lie just beneath the capsule compressing the gland, or be found imbedded in its substance. A hilum-growth may open the kidney and lie encased in the lateral halves; or the kidney may be encompassed by the growth.

The neoplasm may appear as a small, red nodule, the size of a filbert, or as a mass which may attain to one-third the body's weight. In Day's case the tumor weighed 36 pounds; in Mackenzie's it weighed 22 pounds, and the remainder of the child 39 pounds.

The tumor-formation is usually reniform or ovoidal. Intracapsular growths maintain their kidney-shape, even when of large size. Growths that spring from the capsule or hilum may be irregular, even nodular; the same is true of intracapsular growths that have eroded the capsule and bulge through.

The color is generally grayish-white, or translucent. Old tumors that are undergoing degeneration have a dirty, mottled, grayish-yellow appearance; or, if myxomatous in nature, they are clear and gelatinous.

On section the renal neoplasm is very heterogeneous. At first it is encapsulated, but either from rapid increase in bulk or erosion the capsule yields and the growth disseminates. The capsule varies in thickness and is adherent to the tumor, but usually is easily separable from surrounding tissues. A characteristic feature in some is the connective tissue which extends from the capsule into the mass, forming a framework which divides and subdivides the tumor into lobules. In places the growth may be very soft and pulpy from the extravasation of blood; here gelatinous, there cartilaginous, and often throughout cystic in character, the lobules containing either fluid, usually red from admixture with blood, or in old cases cheesy material.

Metastasis occurs late, especially in the congenital form. Only about 50 per cent. of the cases show metastasis. Walker does not think that it occurs through the lymphatics, but by involvement of the renal vein. The lungs are the most frequent seat of secondary growths. In Thornton's case a thrombus was formed in the vena cava and extended into the right auricle. The ureter and bladder are infrequently involved.

The effects and general characteristics of the tumor present the greatest variations, modified by the size and position of the neoplasm. A strikingly alarming feature is the enormous enlargement of the veins, peritoneal and renal, over the tumor. The abdominal viscera are displaced; if on the right side, the liver is pushed upward, the pylorus is compressed, the small intestines are displaced downward and to the opposite side, and the colon occupies a variable relation; it generally lies anterior or to the inner side, or it may cross only the inferior border of the tumor. In left-side growths the colon may lie to the outer side; the spleen is usually pushed upward and backward, capping the tumor; or else in tumors that spring from the upper pole of the left kidney, the spleen may be forced downward and forward.

Classification: Renal sarcomata are classified by Kelynack according to their histological structure as follows: *Round-celled,*

spindle-celled, alveolar, melanotic, adeno-, myo-, angio-, myxo-, lipo-, and *lympho-sarcoma.*

Round-celled Sarcoma: The majority of the growths are a mixture of round and spindle-shaped cells. In very active neoplasms of rapid growth the structure of the tumor is almost homogeneous, consisting of aggregations of round cells in which large and thin-walled blood-vessels or vascular spaces lie. Between the cells there is a scanty amount of fibrillated intercellular substance. The cellular elements greatly predominate. Numerous areas of necrosis are seen. A tumor rich in small round cells, showing a scarcity of large round and spindle-shaped cells, possesses the most rapid growth and greatest malignancy. But few specimens show only one variety of cells.

Spindle-celled Sarcoma: A growth consisting of large and small spindle-cells, with areas of round-celled infiltration, is one of the commonest forms of renal tumor. The predominating feature being the spindle-cells, the growth is so classed. The cells lie interwoven in bundles, separated by an intercellular substance of greater or less density. The round and spindle-shaped cells of these tumors do not show any morphological difference from those ordinarily seen in sarcomata, except in being richer in glycogen.

Rhabdomyosarcoma: In one-fourth of 119 cases in which a careful microscopical examination was made, striped muscular tissue was found (Walker). These rhabdomyosarcomata are peculiar to young subjects. The muscle-cells are elongated and transversely striated, and have no sarcolemma. The striæ are best marked in the larger fibers. The striped muscular fibers lie in bundles disposed in different directions. Unstriped fibers may lie interspersed between the striated, but they are rarely found in any marked amount. Throughout all there is an infiltration of round and spindle-shaped cells in varying proportions.

Angiosarcoma: Many sarcomata are very vascular. Hemorrhagic extravasations account for the rapid and sudden increase in size. There is a distinct variety described as angiosarcoma, with more or less alveolar structure. Harrison thinks that this variety may just as well be called *hemorrhagic carcinoma.*

Adenosarcoma: Adenomatous tissue may appear in round-celled sarcomata, a very different picture from the cystic adenoma or

adenocarcinoma. The sarcomatous elements are interwoven into the glandular tissue of the organ.

2. **Carcinoma:** Bland Sutton says that a large proportion of all renal tumors are in reality sarcomata; but in a few encephaloid tumors of the kidney abnormal development of the epithelium is the distinguishing feature. Reginald Harrison does not attempt to describe the pathological varieties which may occur in this region. Kelynack divides them into two varieties: (1) the true carcinoma springing from the glandular structure, and (2) the epithelioma which originates from surface epithelium.

The glandular forms may be encephaloid, scirrhous, or colloid. The growths that originate in connection with the pelvis of the kidney are epitheliomatous in nature and papillary in form. Harrison says that villous papillomata of the urinary bladder and renal pelvis are not infrequent. Thornton reports a case of papilloma of the renal pelvis containing an imprisoned calculus.

II. Benign Tumors.

Of the benign growths simple *adenomata* are the most common. They appear as solid and cystic adenomata. The cystic form with papillary ingrowth is prone to take on malignant character. Sabourin tells us that they are generally very small, but they may attain to the size of a walnut or an egg. They are situated on the cortex immediately beneath the capsule. Though usually single, the entire kidney may be sprinkled with them. He considers them an accident of renal cirrhosis, and holds that they arise by proliferation of the tubular epithelium. He divides them into two classes, cylindrical-celled and cubical-celled, due to their origin in different periods of degeneration. Grawitz believes that the tubular adenomata arise from proliferation of embryonic fragments of displaced adrenal tissue.

Fibromata, according to Ebstein, are not infrequent, and are found in connection with interstitial nephritis. They are usually small, hard nodules, generally single. Newman refers to several cases that have become of surgical importance through degenerative changes.

Lipomata, myxomata, osteomata, are other forms of benign tumors infrequently met with.

III. Cystic Tumors.

Definition.—In violation of the classical and generally accepted grouping of renal cysts, we have preferred to consider in separate articles Hydronephrosis and Pyonephrosis. Under the head of Cystic Tumors we consider cysts which originate in the cortical substance of the kidney, and also the perirenal cysts of doubtful anatomical origin.

Frequency.—Renal cysts, single and multiple, are frequently met with at necropsy. They constitute an uncommon variety of recognizable renal tumor. They occur most usually as congenital formations.

Etiology.—**Age:** Simple isolated cysts of small size are met with in chronic nephritis, and are frequently seen in subjects of middle and advanced age. Cystic degeneration involving the entire kidney is generally a congenital affection, though not necessarily symptomatically prominent until late in life. It not infrequently attains to great dimensions in the fetus, and we have numerous reports of the tumor so formed acting as an obstruction to delivery.

Sex: There are no conclusive data showing preference for either sex.

Simple cysts resulting from an accumulation and retention of secretion, due to occlusion of a uriniferous tubule, or at the mouth of a capsule of Bowman, are commonly met, associated with and dependent upon interstitial nephritis. They are true retention-cysts.

Following the teaching of Virchow, pathologists generally consider *multiple cystic degeneration,* that is, *fetal cystic kidney,* due to obstruction at the papillæ, in consequence of fetal renal inflammation, or to irritation and obstruction of the tubules by urates. Köster assumes that the primary lesion is in the lower urinary tract. Bland Sutton says: "The only tumor of the kidney to which the term renal adenoma is applicable is that peculiar condition known as congenital cystic kidney." Ellermann likewise considers them due to actively proliferating epithelium.

The histogenesis of *adult cystic kidney* may be regarded as undetermined. Authorities generally hold that the *acquired cysts,* that is, those that appear in adult life, are similar in origin to the

PLATE XVI.

KIDNEY. CONGENITAL CYSTIC DISEASE.

U. Ureter.

Surface of organ.

(*Two-thirds natural size.*)

A section of this kidney is shown in Plate XVII.

simple cysts, and are a result of interstitial renal fibrosis. Ritchie, whose observations extend over 88 cases, concludes that cystic degeneration of the adult kidney is a kind of adenoma. This is in accord with Bland Sutton, as expressed above. Shattuck, and Still regard them as a result of developmental error, whereby the mesonephros has blended with the metanephros, and the cysts originate not in the proper uriniferous tubules, but in the Wolffian body. Presuming this view to be correct, Dickinson and Fenton claim that it explains the progressive and ultimately fatal nature of cystic kidney, on the ground of the well-known tendency of organs which are congenitally malformed to become diseased. The frequent bilateral occurrence of the disease, and a similar cystic degeneration in other organs, the liver especially, would suggest a general cause; probably the theory of embryonal inclusion is correct.

The **cystadenoma,** occurring in adult life, and which Kelynack regards as malignant, is not infrequently bilateral, and like the congenital form has similar processes in other organs. If not congenital, it is of unknown origin.

Parasitic cysts are usually due to the tenia echinococcus.

PATHOLOGY.—1. **Simple Cysts:** The ordinary simple cysts that occur singly or in numbers as the result of generalized sclerosis are usually situated in the zone between the medulla and cortex. Their capsules are the condensed connective tissue of the kidney. The intervening tubules may be healthy. These simple cysts may occur in a kidney otherwise healthy, and also as complications of other conditions. They are of small size, and rarely attain to surgical importance.

2. **Congenital Cysts:** This multiple cystic formation usually involves both kidneys and the entire gland, though either pole or half may be affected. The cysts vary in size, and are entirely distinct from each other. The secreting structure of the kidney is attenuated or destroyed. Not infrequently other malformations are found.

3. **Acquired Cysts** are numerous and of varying size. The walls are thin and enclose finger-like projections covered with epithelium. Not infrequently a glomerulus is discovered within the cyst. Its fluid contents show an admixture of epithelium, blood, and detritus. Portions of the kidney may retain secret-

ing structure, though compressed, and there is abundant proliferation of connective tissue. The bilateral character of the disease, the age at which it occurs, usually about forty-five years, and similar processes in other organs are features which indicate its neoplastic nature.

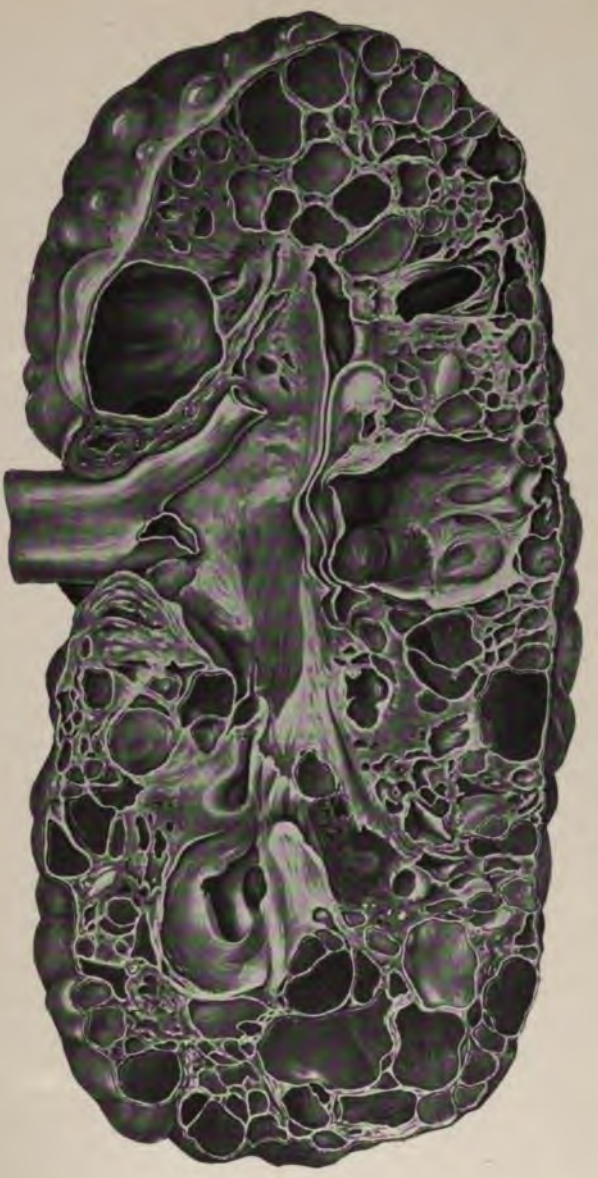
4. In **cystadenomata** the cystic expansions are usually not larger than an ordinary tubule; others may attain to considerable size. Each cyst is lined with a layer of cylindrical epithelium, and this form is especially prone to develop papillary ingrowths; Cherry and Allen excellently describe this type, which Reginald Harrison thinks is probably hemorrhagic carcinoma.

5. **Echinococcus cysts** of the kidney may form large fluctuating tumors of slow growth, gradually destroying the kidney-substance by pressure. The contents contain the scolices with their hooklets; or the translucent, thin-walled vesicles may be found entire. Sterile cysts may be recognized by the characteristic lamination of the walls. They may rupture and spontaneously disappear by discharge of their contents through the ureter; but perforation into adjacent organs or cavities is more common.

6. **Paranephric cysts** are of very doubtful origin. There is no evidence in the kidney that they are of cortical growth. They originate either from the capsule or from the lymphatics. They rest upon and compress the kidney. Abbe records two examples of this form of cyst, producing large abdominal tumors.

Suppurative processes developing in cystic kidney are exceedingly rare; Mohr refers to only 3 such cases. Farr operated for kidney-tumor, the patient presenting malarial symptoms, and found cystic kidney with pus-like fluid which proved to be sterile. Bardenheuer extirpated a purulent cystic kidney which developed in the course of general septic disease; the patient recovered.

Symptoms and Physical Signs of Renal Tumors.—**History**: In reviewing the clinical history of renal growths but small importance can be attached to any special cause. Neither traumatism, previous disease, nor calculous conditions occur sufficiently often to assume definite etiological prominence. Heredity has a very vague influence in malignant disease; there are only 8 cases in Walker's 142 that could be ascribed to such descent. Steiner demonstrates in a remarkable manner the influence of heredity in adult renal cysts by giving two genealogical tables in



KIDNEY.

CONGENITAL CYSTIC DISEASE.

Section of Organ.

(*Two-thirds natural size.*)

The surface of this kidney is shown in Plate XVI.

which several members of the same family show cystic kidney. There are two periods in life in which renal growths are prone to develop. In childhood, massive, rapidly-growing, unilateral abdominal tumors, are in all probability renal sarcomata; bilateral tumors, noticeable soon after birth, are probably cystic; young adults are infrequent subjects of renal enlargements. The polycystic kidney is most common in middle age. All tumors may pursue a latent course, their existence unsuspected until the enlargement becomes conspicuous. But few patients, however, escape urinary disorders. Though the renal substance is much destroyed by disease, whether cystic or solid, polyuria is a frequent symptom. We can often obtain the history of indefinite pains, polyuria, and hematuria. In adult cystic degeneration slight alterations in the urine may be the only symptom, and the patient succumbs to uremia, the autopsy showing bilateral degeneration with complete destruction of renal substance without appreciable enlargement. The diagnosis of renal tumors rests upon the history of urinary disorders and the development of a tumor; the other symptoms are common to abdominal growths.

General Condition of the Patient: All massive tumors affect the general condition of the patient, as is shown by anemia and loss of flesh. Malignant tumors rapidly develop a cachexia, which is especially characteristic in children, appearing much earlier in them than in adults. It seems that cachexia, however, is more pronounced in adults suffering from carcinoma than in sarcoma.

Pain: In infantile sarcoma pain is present in about one-third of the cases. Carcinoma, owing to its complications, gives rise to more pain. Cystic degeneration may exist painlessly. Pain is by no means a constant or diagnostic symptom. It may occur paroxysmally in all forms of tumor, associated with hematuria. The weight and traction of the tumor produce backache and a dragging, tired sensation. By pressure a tumor may cause sacral or crural pain. The encroachment of the tumor upon the abdomen often excites localized peritonitis, from which the patient may suffer. Much discomfort is attributable to visceral compression and displacement.

Tumor: A recognizable tumor-mass is the most reliable physical sign in all neoplastic formations and cystic degenerative processes in the kidney. It is often the earliest, and frequently the only symptom. Of 142 cases of sarcomata in children analyzed by Walker, tumor was absent in only 2. In individuals of all ages, of 133 cases of renal neoplasms Guillet records that in all but 4 there was a recognizable tumor. Ebstein found tumor absent in only 3 out of 52 cases. And in 100 cases of sarcomata in children Chevalier found it the initial symptom in 60 per cent.

In cystic degeneration it is not so constant a sign as in solid tumors. Out of 88 cases studied by Ritchie, 21 only presented a recognizable tumor during life. Of 63 cases of hydatids, in 18 there was a palpable tumor.

Israel asserts that all enlargements the size of a walnut should be palpable; he prefers the lateral decubitus. By Guyon's method, with patient in dorsal position and limbs flexed, with one hand resting lightly upon the abdomen, the other is pushed under the lumbar region; and with this hand the operator makes gentle but quick blows which project the kidney in a jerky manner toward the abdominal wall, *ballottement.* Tumors of moderate size may thus be located. Obese subjects offer difficulties to examination. Small tumors located in the upper pole of the kidney may escape detection. Renal growths primarily occupy the lumbar region. Tumors developing in movable kidneys may appear in the epigastrium (Ritchie), but usually in the umbilical or hypogastric regions. They occupy the costo-iliac space, and develop forward, rarely producing any bulging in the loin, merely effacing the natural curvature. Large cystic tumors may occupy a central position (Abbe). Any growth that attains to enormous dimensions may practically occupy the entire abdomen, but the history of its development will indicate its site of origin. Small tumors are mobile under palpation, and sometimes respond to respiration, and may be influenced in their position by change of attitude of the patient. Large tumors are immovable.

In contour it was originally stated by Jenner, and emphasized by Morris, that all renal tumors are reniform. This characteristic shape is maintained in all disseminated intracapsular growths, and it is very proper to describe a renal tumor as ovoid, oblong,

or kidney-shaped. There are numerous exceptions to this, as the pathology would indicate; and while the borders are generally rounded, irregularities in outline are met with. Infantile sarcoma is characteristically smooth over its surface, ovoidal in shape, with rounded borders. Cystic tumors are nodular, irregular, but taken *en masse* usually maintain the reniform outline. Irregularities are due often to adherent omentum, to displaced and attached viscera.

The palpating fingers may be inserted between the tumor and the liver. Very rarely does the growth extend into the true pelvis; deep abdominal pressure will define the lower border of the tumor. The posterior border cannot be defined; it lies in close relation with the lumbar muscles.

Rapidly growing sarcomata and carcinomata are soft, boggy or elastic; older ones, undergoing degeneration, may be fluctuant; those of slow growth are usually firm, even hard. A cystic kidney is knobby, and as a whole does not show fluctuation; areas of fluctuation may be found. Parasitic and paranephric cysts are widely fluctuant.

Tenderness under palpation is not present unless there is some complicating inflammation.

A renal tumor under percussion shows dulness externally and posteriorly; there is no area of resonance between the tumor and the spine. Careful examination with the patient standing will usually reveal a zone of resonance between the tumor and the liver. Large tumors reveal dulness over their anterior surface generally; the percussion-note obtained over the abdominal aspect depends upon the relation of the intestines to the tumor, and this, in turn, varies with the length of the mesocolon, the size of the tumor, and the mobility of the kidney. It may be asserted that some portion of the anterior surface of a renal tumor is covered by the colon. Right-side growths force the colon inward, and downward usually, so that it is found, as is evidenced upon percussion, skirting the inner border, or crossing the anterior-inferior surface, exceptionally along the upper and inner border. Kelynack illustrates an instance in which a large tumor of the left kidney had advanced under the colon, and the intestine lay along the outer surface. Pararenal cysts produce displacement of the colon outward; 3 such cases are re-

ferred to by Pawlik. Very large growths may be covered by small intestines, and very frequently by omentum. In defining the relation of the colon rectal insufflation with gas will be of greatest aid. Auscultatory percussion should also be employed.

Occasionally a soft, blowing, systolic murmur is heard over a renal tumor.

Glandular Involvement and **Metastasis**: These are late complications in malignant tumors of the kidney, and rarely recognized during life.

Varicocele: Some attention has been given to the occurrence of a symptomatic varicocele. It is of especial significance when it occurs on the right side. It is regarded by Hauser as evincing glandular involvement.

Pressure-effects: Tumors of moderate size, especially those developing in movable kidneys, produce constipation and digestive disorders. Large tumors of the left side may displace the stomach; those of the right side compress the duodenum, giving rise to symptoms of pyloric obstruction and gastric dilatation, though tumors of enormous dimensions may in no way disturb the functions of the alimentary canal. The more direct effects of pressure are seen in edema, occasionally in ascites and enlargement of the surface veins.

The Other Kidney: Many of the benign growths occur simultaneously in both kidneys. Primary malignant disease has never been known to occur bilaterally; should the other kidney become involved, it is by metastasis. Of special importance is the knowledge that cystic degeneration, both in infants and in adults, is generally a bilateral affection; all authorities emphasize this as a classical sign. This phenomenon does not necessarily appear simultaneously in each, yet Albarran, and Bazy impress us that a recognized cystic degeneration in one kidney should imply a similar process in the other.

The Liver: All large kidney-tumors of the right side may displace the liver upward, or, acquiring adhesions to it, may drag it downward. By direct pressure or traction the biliary channels are involved, and jaundice is not an infrequent symptom. In polycystic kidneys not infrequently there is enlargement of the liver with areas of cystic degeneration, and this should be

conclusive in its diagnostic significance. Lejars found this complication in 18 out of 63 cases.

The Heart: Cardiac hypertrophy is a common complication of renal cystic degeneration.

The Urine: Albumin, either in trace or in high percentage, occurs in the urine especially in cystic kidneys; it is an inconstant symptom in malignant neoplasms. Tube-casts and epithelia are occasionally found. In a few cases of fungating sarcoma or carcinoma, portions of the tumor may be discharged and microscopically recognized. Hematuria, after tumor, is the most important and diagnostic sign of renal growths; it is a very constant sign in cystic kidney, and occurs in about 40 per cent. of all malignant tumors; it is more frequent in sarcoma than in carcinoma. The color of the urine varies with the persistency and quantity of admixed blood. It is usually of a dark-brown or smoky color, occasionally bright-red; it may be black from melanin. Fresh and copious hemorrhage may contain ureteral casts, generally considered pathognomonic of renal hemorrhage; it is by no means diagnostic of renal tumor. There are many conditions of the kidney in which hematuria is a symptom, but when under microscopic examination there is a characteristic rosette-like structure, arranged in concentric or radiating form, it is considered a pathognomonic sign of cystic kidney; unfortunately it occurs as an intermittent symptom (Höhne, Jacob and Davidsohn). Blood from malignant renal tumor will often show in the urine as yellowish-gray or granular matter; if microscopically examined, round and spindle-shaped cells may be found. The detection of hooklets in the urine affords the only positive diagnosis of hydatids, without puncture; sometimes daughter-cysts are discovered.

Exploratory Puncture and **Incision:** For diagnostic purposes we are warranted in aspirating an elastic or fluctuant area in supposed cystic kidney. The result may show clear fluid, which is negative; but if the brownish-red fluid shows rosettes, the diagnosis is positive. If in face of all evidence the diagnosis is still doubtful, direct examination of the kidney through lumbar incision is warranted.

DIFFERENTIAL DIAGNOSIS.—The three symptoms, tumor, hematuria, and cachexia, form the basis of the diagnosis of renal tu-

mors. Having determined by topographical landmarks that the swelling belongs to the kidney, hematuria and cachexia point to its malignant nature. Most difficult of diagnosis are those cases in which a tumor occurs unassociated with either of the other symptoms, or in which hematuria appears in the absence of a tumor. A tumor is usually the first sign in congenital sarcoma; an intermittent hematuria, the leading one in cystic degeneration; hematuria and cachexia may both be present before there is a tumor in carcinoma. The impression must not be gained, however, that cachexia is a conspicuous phenomenon in renal neoplasms; the anemia and emaciation are generally marked in all abdominal growths as an effect of pressure. Surgically speaking, it is not so important to determine the character of growth, were it not that cystic kidney is so frequently a bilateral condition. Instances of simultaneous development in both kidneys with bilateral tumor are very rare. We must, therefore, rely upon the urinary analysis to determine the condition of the other kidney.

Renal Tuberculosis: The greatest difficulty is encountered in differentiating small neoplasms from tubercular lesions in the early stage. Pain, tenderness, and vesical disorders, even polyuria, may be common to both. The diagnosis can only be made through microscopical examination of the segregated urines.

Calculous Pyonephrosis: Perhaps the most common error is the mistaking of cystically degenerated kidney for calculous pyonephrosis. The latter condition is only exceptionally bilateral; the other symptoms are very similar, though the hematuria in calculous conditions is usually microscopical in quantity, and excited by exercise or manipulation. The septic phenomena do not occur in cysts.

Stable Hydronephrosis: Parasitic and paranephric cysts cannot be differentiated from stable hydronephrosis except by sounding of the ureter, determining the patency of that duct.

Malignant growths in the *retroperitoneal glands* closely resemble renal tumors. They often yield the same physical signs, cause the same urinary disturbance, and are attended by cachexia. The chief points of distinction are: the glandular tumors are very rare, more central in position, the colon usually to the outer side, and there is an absence of hematuria.

Enlarged spleen is not common in children, its edges are sharp and notched; and unlike renal tumors, it is not covered by intestines. Of greater significance, however, is the presence of a tympanitic area between the tumor and flank in splenic enlargements.

PROGNOSIS.—*Sarcoma* in children progresses rapidly; in a few months it may attain to enormous growth. The average duration of life from its inception is about seven months. In adults the duration may be two or three years.

Carcinoma is slower in development. Cases are reported in which it has remained latent several years; the average duration of life is about two years.

Congenital cysts often cause death *in utero* or in delivery. In adults cystic degeneration may exist for many years; death is usually due to uremia, or to pressure.

CONTRAINDICATIONS TO OPERATION.—Confronted with the statistics of Walker, the latest and most complete, of the results of nephrectomy for sarcoma in children, which show an immediate mortality of 38.25 per cent., absolute cures of 5.47 per cent., the others dying from recurrence usually within a few months, the question may be well asked, Is nephrectomy for malignant disease in children justifiable? On the affirmative side we find Israel, Abbe, Tuffier, Kelynack, Albarran, in fact all operators of greatest experience; Osler considers it justifiable in selected cases. That the operation may prolong life cannot be denied; that an absolute cure may follow in very occasional cases is true; yet, though admitting these points, it must be regarded as a questionable procedure, justified only in the early stage of the development of the disease, and by the findings after the exploratory incision.

In malignant disease in adults the mortality is not so great. At the Moscow Congress, Israel claimed that 6 of his 24 cases showed no recurrence after 3 years, and that his primary mortality was 20 per cent. Küster gives the operative mortality of 41 per cent. in 263 collected cases, yet he could find only 9 in which there was no recurrence after three years. Butlin holds that the results do not justify the continued trial of nephrectomy for carcinoma. Barth has collected the statistics of 100 nephrectomies performed for malignant disease up to 1892; of these 42

died from operation, 20 from metastases; the remaining 38 were reported as cured, the duration of the cure not given. Rovsing states that nephrectomies for malignant tumors prior to 1890 gave a primary mortality of 50 per cent.; since then, a mortality of 25 per cent. Though not encouraging, if the contraindications of pronounced cachexia and metastasis can be excluded, the results in certain cases seem to justify the operative treatment.

The well-known tendency of congenital and acquired cysts to form in both kidneys is sufficient reason to discourage operative treatment of this condition. Of 25 published operations for multiple cysts, 22 were nephrectomies, with 7 immediate deaths. In following the after history of many of these patients who recovered from operation, we find that several died in short periods from anuria, indicating an unrecognized degenerative change in the other kidney. The constancy of this condition interdicts nephrectomy for polycystic kidney unless by the most careful investigation (ureteral catheterization and even exploratory incision, if needed) one kidney is shown to be healthy.

Surgical Indications.—Simple renal cysts of moderate size demanding operation may be drained after *nephrotomy*, or may be excised.

In polycystic kidney, when neither organ is sufficiently sound to justify removal of the other, the walls of the larger cysts may be incised to reduce the size of the organs and the kidneys then stitched to the lumbar muscles. This has been done successfully by Curtis, by Kammerer and by Lund.

Parasitic cysts should be treated, when feasible, by *partial nephrectomy*, with excision of the germinative membrane; otherwise, by incision and drainage. Of 20 cases collected by Howzel, in 6 *nephrectomy* was done, with 5 deaths; of 14 treated by simple drainage, all recovered.

All innocent tumors that attain sufficient size to demand interference should be removed, if possible, by *partial nephrectomy*.

Malignant tumors demand removal by *nephrectomy* on account of pain, hemorrhage, and pressure-symptoms. But the contraindications must be well weighed. Abbe considers it good surgery to do a *partial nephrectomy*. Moynihan recently records the removal of a myxosarcoma by amputation of the lower half of the kidney.

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HEMATURIA.

Blood in the urine appears as a symptom of many well-recognized morbid conditions; but surgeons and internists are often perplexed by persistent, painless and depleting hematuria, in which no demonstrable lesion can be determined. This symptomless hemorrhage was attributed by Senator to a constitutional condition, and described by him as "renal hemophilia"; yet he admits that subjects of it are uniformly cured by operation. Klemperer describes such cases as "hematuria from a healthy kidney," and considers it an angioneurosis. He asserts that they can be cured without operative treatment. Harrison, in his Presidential Address in 1896, calls attention to the frequency of hemorrhage due to renal tension. Rovsing, Newman, and others report cases, and remark upon the frequency of hematuria in movable kidney. Fenwick says that in some of these cases of hemorrhage from a single kidney it emanates from varicosity of the papillary plexus, and that the site of the disease is apparent only upon opening the renal pelvis; the removal of this vascular papilla will control the bleeding. Keys regards chronic nephritis as the common cause of renal hemorrhage. The consensus of opinion at the French Congress, in 1900, was that the so-called angioneurotic bleeding does not exist, and that hematuria from a single kidney, which is now easily determined by cystoscopic examination, is an evidence of a definite, local, morbid process.

The treatment of such cases is the incision ordinarily employed for nephrotomy, or directly into the renal pelvis, as Fenwick advocates. The operation often fails to demonstrate a specific lesion, but is almost uniformly curative in its effect.

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CHRONIC BRIGHT'S DISEASE.

In 1896, Reginald Harrison chose as the subject of his Presidential Address to the Medical Society of London the "Treatment of Some Forms of Albuminuria by Reni-puncture"; but it remained for George M. Edebohls to develop and successfully apply a special surgical procedure as a curative measure in advanced forms of chronic Bright's disease. He first publicly proposed his measure in the Medical News, April 22, 1899, though his observations began as early as 1892. In the Medical Record of December 21, 1901, he reports 18 cases of chronic Bright's disease treated surgically. He advocates freeing the kidney from its fatty capsule, then delivery of the kidney, and division of its proper capsule along the entire length of the convex external border of the organ. Each half of the capsule is then stripped from the kidney and reflected toward its pelvis, where it is left. The raw kidney is now dropped back into its fatty bed, and the external incision closed without drainage.

The theory advanced to account for the favorable change is that, by the formation of adhesions between the kidney-substance and adjacent tissue, many new blood-vessels are soon added, whose blood is directed to the kidney-tissue, and thus hyperemization of the organ results, initiating a gradual and progressive cure, accomplishing something more than the relief of renal tension claimed by Harrison.

There was no operative mortality in his series of 18 cases. 8 patients, a year or more having elapsed since operation, remained cured; "they are free from all symptoms referable to the kidneys." These are most remarkable results, and justify a most serious consideration by the surgeon.

A. H. Ferguson reports 2 cases in which he employed practically the same procedure, with puncture of the kidney-substance; both patients have been relieved.

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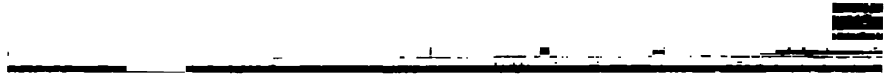

ADRENAL GROWTHS.

DEFINITION.—Under this head we consider neoplasms developing from the suprarenal bodies, or derived from accessory outlying bits of the glands.

FREQUENCY.—Roger Williams found only one primary adrenal growth in 8,378 cases of malignant disease. In 1898, Rolleston and Marks, limiting their collection of cases to those of undoubted malignant disease, were able to collect 26 cases. Ramsay, who was less critical in his selection of cases, presents a study of 67 cases of primary malignant tumors of the suprarenal glands.

ETIOLOGY.—**Age:** Unlike renal growths proper, they are more common in middle life. Rolleston and Marks give the average age at thirty-seven years; Affleck and Leith at forty-five years. Pepper recently reported a congenital sarcoma of the liver and suprarenal in a child six and a half weeks old, and found 5 other similar cases. In Ramsay's series the youngest was only nine months old, while the oldest was seventy-three years.

Sex: There is no special predilection for either sex.

Any effort to discuss the histogenesis of these growths would be merely a rehearsal of the well-known theories of Cohnheim, Grawitz, and McWeeny.

PATHOLOGY.—The primary enlargement may be purely a glandular proliferation, called "adrenal goiter." The tumors are either adenomata, sarcomata, or carcinomata. Adenomata, according to Rolleston, may be single or multiple; and the greatest difficulty is encountered in distinguishing them from adenocarcinomata. Of the malignant tumors, sarcoma is far the most common; some authorities even deny the existence of carcinoma. Rolleston and Marks found 9 malignant adenomata or carcinomata in their series of 26 cases. All malignant growths are very vascular, soft, and of rapid development, and show a tendency to degeneration. They may spread entirely subperitoneally, invade the right lobe of the liver, involve the kidney, or extend into the suprarenal veins and vena cava. Secondary growths occur most

frequently in the liver and kidney. The lungs, bronchial glands, and pleuræ may all be involved.

Symptoms.—The general symptoms of adrenal growths imitate very closely those of renal neoplasms; even hematuria may occur, which increases the difficulty of diagnosis. Adrenal tumors are usually of small size and of rapid growth. They may occasion symptoms suggestive of Addison's disease, discoloration of the skin, an abundant growth of hair, and marked cretinoid appearance. Analytical studies, however, show that the clinical picture of Addison's disease is not presented. Marked asthenia, accompanied by languor, was noted in 22 patients (Ramsay). Digestive disturbances are not uncommon, and may be accounted for by peritoneal invasion or mechanical pressure. Dyspnea, uneasiness, and pain are symptoms attributable to the presence of the tumor.

A tumor may be palpable, situated in either hypogastric or lumbar region. It is usually quite movable, may be pulsatile; it closely resembled an abdominal aneurism in Rolleston's case.

Differential Diagnosis.—The small, movable, right-side tumors present the greatest difficulties in differentiation from distended gall-bladder. The history of biliary colic or obstruction, and the superficial position of the gall-bladder would distinguish it from an adrenal growth, which is comparatively painless and underlies the intestines.

Rolleston seems to think that there would be some difficulty in distinguishing these tumors from a pancreatic cyst. The position of the latter, its tense, elastic feel, and relation to the stomach characterize it.

The common error is to mistake an adrenal tumor for a renal enlargement; that this differentiation cannot often be made in the ablated specimen either macro- or microscopically excuses the clinical error. The extreme mobility of the tumor, the absence of urinary symptoms, and the fact that adrenal tumors are common in middle life, while renal ones are common in infancy, are aids to diagnosis. Morris asserts that the diagnosis has been correctly made in only two instances.

Prognosis.—The course of malignant disease in the adrenals is very rapid; the duration is rarely longer than six months. Secondary growths are very common, especially in the liver. In 17

of Ramsay's 67 cases the disease was bilateral. In carcinomata metastases are more common than in sarcomata.

SURGICAL INDICATIONS.—The only hope for life lies in an early operation. Metastases, adhesions, and hemorrhage may render this difficult or impossible. Kelly's first case was very simple; in his second, operation was incomplete. Roberts was compelled to abandon his operation because of hemorrhage.

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PENETRATING GUNSHOT WOUNDS OF THE ABDOMEN.

DEFINITION.—The limitations of this subject are restricted to a consideration of wounds that have entered the peritoneal cavity, with or without visceral injury, produced by missiles propelled by the explosion of gunpowder.

FREQUENCY.—Penetrating wounds of the abdomen constitute about 6.6 per cent. of all wounds coming under treatment (Serrier). Otis's percentage is 4.28 from field hospitals, which is naturally higher than in fixed hospitals, since many die before being removed to the latter. In civil practice, it seems safe to make the statement, without authoritative statistics, that wounds of the abdomen occur in greater relative frequency; the close range and accurate aim account for this. The pistol-carrying habit in the South and West is responsible for the greater number of cases reported from these sections, the individual experience of several southern surgeons being most extensive. From January, 1892, to January, 1901, inclusive, laparotomy was performed in the Charity Hospital in New Orleans, 113 times for gunshot wounds of the abdomen (Fenner).

GENERAL PATHOLOGY.—A missile may reach the abdominal cavity directly through its walls or from some remote wound of entrance. When it has passed through the parietal peritoneum it becomes a *penetrating* wound. If in its course through the abdomen any of the viscera, mesentery, or omentum are wounded, we then designate it a *perforating* wound. It would be preferable to limit the term 'perforating' exclusively to wounds involving the entire thickness of the wall of a hollow viscus, and describing the wounds of the solid viscera as incised, punctured, lacerated, etc. Yet we shall use the term 'perforating' in its commonly accepted sense.

The wound of entrance is usually situated on the anterior or lateral surface of the abdomen. A projectile entering the back meets with more resistance, may be arrested or deflected by the

ribs or vertebræ, yet it may pursue a direct course straight into the abdomen; such wounds are most difficult in their diagnosis, and most serious in their effects. The penetrating power of missiles from modern firearms renders every part of the body in the line of direction of the missile vulnerable; Senn refers to a number of cases of gunshot wounds of the abdomen, the wound of entrance being in the neck or chest. In wounds of the thorax which also penetrate the abdomen, the latter complication is frequently overlooked because of failure to appreciate the arch of the diaphragm and the relative anatomy. Wounds of the buttocks or thigh may by their course become abdominal. In Case V of my series, the bullet entered the buttock of the right side, passed through the greater sciatic notch, produced thirteen intestinal perforations, and lodged in the muscle directly opposite the point of entrance. The remote location of the wound of entrance does not, therefore, eliminate the possibility of abdominal penetration.

The appearance of the wound of entrance is influenced by a number of factors. The rifle used in modern warfare, with its nickel-clad bullet, has enormous penetrating power, and produces a very small tubular wound of entrance, perhaps with a narrow surrounding zone of ecchymosis in the contused tissue. In civil practice the wound is often large, ragged, powder-burned, containing bits of clothing and wadding. The angle of entrance may sometimes be observable from the obliquity and staining of the wound. It is exceptional for much bleeding to appear from the wound of entrance. In ordinary bullet wounds it is unusual to have protrusion of the viscera, though occasionally the omentum may be extruded; but in larger wounds produced by shell, there may be protrusion of viscera. In Case XVII of my series, a negro girl was shot with a toy cannon; in this case paper and rags were the missile, making a large wound of entrance, two inches in diameter, just to the right and below the umbilicus. Through this there was hanging some eight or ten inches of small intestine torn from its mesentery and riddled by the wadding.

The peritoneal wound does not always lie directly beneath the surface wound of entrance. The angle of impact determines the length of the mural track; the tendency of all bullets is to pursue

a straight course. Serious consequences may attend non-penetrating wounds in the abdominal wall.

After penetration it is said that visceral injury follows in 97 per cent. of cases. Stimson records two cases of penetration without perforation. Senn reports two cases out of sixteen in which the hydrogen-gas test showed no perforation, and recovery without operation followed; he also reports four out of sixteen without visceral lesion in experiments on the cadaver. Hunter McGuire reported four undoubted cases of this character. Oliver finds three instances of penetration from a .44-caliber ball fired at close range; the viscera escaped injury; in each of his cases the diagnosis was made by laparotomy, and all recovered. Finkelstein notes the comparative infrequency of complications of abdominal wounds by wounds of the hollow viscera. In 132 cases 70.4 per cent. proved to be merely penetrating wounds of the abdominal wall. The reports of the numerous recoveries after abdominal wounds in the late wars in Cuba, the Philippines and Africa, in which the missile employed was the small steel bullet, indicate that hollow viscera were not injured. Instances might be multiplied, but the above are sufficient to show that penetration is not always followed by perforation of viscera. From experiments and clinical observations it appears that in wounds above the umbilicus the viscera may escape; but there is great danger in such wounds of injury to the vascular roots. In wounds to the right or to the left of the umbilicus with antero-posterior course the viscera may escape. But in wounds below the umbilicus and in those from side to side, as also in all instances in which the range is oblique, the viscera are almost invariably wounded. Too much emphasis has been laid upon the probable harmlessness of wounds above the umbilicus; I have found them invariably perforating in character. Let it be said and understood that perforation is to be inferred after penetration of the abdomen in every instance.

Owing to the great length, manifold convolutions, and space occupied, the small intestine is more frequently wounded than other viscera. In order of frequency would next occur the liver, stomach, large intestine, kidneys, spleen, and pancreas.

The course that a bullet pursues after it enters the abdominal cavity is in the direct line of its projection. That viscera are

wounded which occupy positions distant from the line of transit of the bullet is explained, first, by the change in the posture of the patient from the position at the time of injury to that assumed at the time of examination; second, by the natural mobility of certain organs; third, by the change in the form and position from the inherent contractility of the viscera. Parkes contends that the ball may pursue an erratic course, deviating from its line of flight, when its "velocity and power of penetration bear such a relation to the power of resistance of the different tissues in the abdominal wall and contents, that the softest of these, touched in a certain way, will deflect its course." My clinical experience and numerous experiments upon dogs do not incline me to accept the conclusion of Parkes, but in the language of McGraw my views are well expressed: "I am very much inclined to the belief that the whole doctrine of balls glancing from soft tissues is a fable, founded upon imperfect observation and wrong deductions."

The character of the visceral wound, and the damage done by the ball in perforating wounds, depend upon a number of correlating causes. In a general way it is true that the larger the missile with its proportionate velocity and energy, the greater its destroying power, and the more serious its consequences. In Case XXI of my series, a derringer ball of .56 caliber completely severed the gut—an unusual injury that could scarce have been inflicted by a ball of small caliber. Of more consequence than the size of the bullet, however, is the angle of impact. The size of the wound depends upon whether the course of the bullet is transverse, oblique, or parallel to the axis of the gut. In Case VI a .22 flobert caused a linear wound one and a half inches in length along the mesenteric border. The larger the bullet the greater the likelihood of visceral injury, and the smaller the bullet the less such probability. In wounds of hollow viscera the size of the wound depends also upon the amount of fluid they contain at the time of the accident. The distance of range is not as important a factor in determining the character of the wound in the abdomen as in other portions of the body. Lacerations from the explosive effect upon the solid viscera is enormous at short range, and is still manifest at 800 to 1,000 meters. A clean perforation of the liver, spleen or kidney can hardly be said to occur

at any range (Forwood). The lateral energy of a bullet shows its effects in the destruction or disorganization of the glandular organs, often fragmenting them. The wound of entrance into these organs is often stellate. Influenced, then, as the wounds are, we may find the alimentary canal simply perforated; a round hole; obliquely cut; severed entirely; or exhibiting a linear gaping wound. The wound of solid viscera, though often insignificantly small in appearance, is deceptive as to the extent of injury that has been wrought.

A perforation of the small intestine is followed by an extrusion of the mucous membrane; it appears like a rosette or prolapsed rectum. This effect is caused, first, by the redundance of mucous membrane over the serous coat; and second, by the spasm of the circular fibers of the gut. In very small perforations this will not occur, nor is it so noticeable in long, nor in very oblique wounds. I have not seen it in wounds of the stomach or colon; and to this fact may be attributed the possibility of spontaneous healing in small perforations of these viscera. Wounds of the small intestine do not spontaneously heal, and if the patient lives a fecal fistula may form.

Wounds of the intestines do not bleed very freely. Mesenteric, omental, and wounds of solid viscera may all bleed copiously. Hemorrhage is the most important primary phenomenon of visceral injury. Abdominal blood-vessels of insignificant size when severed, under the influence of intra-abdominal pressure, bleed continuously. The shock which attends peritoneal wounds, as is well known, causes visceral engorgement, and this encourages the hemorrhage.

The question of extravasation of the contents of the viscus is of the greatest pathologic import. Stomach-wounds, when sufficiently large, are usually followed by immediate extravasation of stomach-contents. Wounds of the gall and urinary bladders permit a complete discharge of their contents, which may appear at the external wound. In wounds involving the intestines it is the almost universal statement of text-books that extravasation takes place immediately upon the receipt of the wound. This statement is not borne out by the experiments of Parkes, Schachner, and others, nor by clinical observation. Extravasation does take place, it is true, but Schachner makes the point, When?

Usually not until the intestines are handled, or until the gut is distended, opening the wound, which is generally after twenty-four hours. The immediate extravasation, even in extensive wounds, is far less than one inexperienced would suppose. In the case previously cited in which the gut was severed entirely, there was no free fecal matter or intestinal contents found. Murphy very clearly states that extravasation of fecal matter is caused by manipulation. The ectropion of the mucous membrane, the valve-like character of the wound, and the arrested peristalsis are the chief agents in preventing extravasation. If peristalsis is resumed the mucous membrane is drawn inward, and extravasaton will occur; and as above stated, the gaseous distention of the intestines permits the escape of contents. Yet no perforation, however small, can be relied upon to prevent the escape of infectious gases and contents. From the smallest perforation fatal septic peritonitis may arise. For a time extravasation may be limited by inflammatory adhesions; but the supply of infectious matter is generally too great to be so localized.

Apropos of the question of extravasation, the effect of a gunshot wound upon the intestines is for a time to suspend or inhibit its peristalsis; this is clinically recognized by the arrest of bowel-action. This effect is secondary to a primary spasm.

Wounds of the mesentery, involving a mesenteric artery or vein, are of pathologic and prognostic significance. The copious hemorrhage that attends them may result in immediate death. The remote effect is the liability to gangrene of that portion of the gut supplied. Keen records a case with multiple and complicated wounds, in which he attributed death to a wound of the superior mesenteric vein. Small wounds close to the mesenteric border of the intestines, as Schachner has shown, are liable to be followed by gangrene.

The effects of any given penetrating wound of the abdomen are problematical. Their nature is uncertain; the tract is always septic; the number of perforations unknown; complications can not be foretold. If a ball of average velocity enters the abdominal cavity, perforation is to be expected. That it may find its way between the interstices of the intestines we have previously admitted as a possibility; but that it may produce perforations of the intestines, from one to twenty-eight in number, is a recorded

fact; that, if solid viscera lie in its course it will traverse these and, in all probability, will wound other viscera. Multiple perforations occur most frequently in the ileum, and are oftenest made by bullets that pass through the abdomen obliquely from side to side.

The above may be taken as the immediate pathological effects of penetrating and perforating wounds of the abdomen. The subsequent effects depend upon the nature of the injury, the amount of immediate or recurring hemorrhage, the viscera injured, the presence of extraneous material—that introduced by the missile, the escaping intestinal gases, and extravasated visceral contents. Simple penetrating non-perforating wounds may be attended by hemorrhage. If this hemorrhage is small in amount and the absorbing power of the peritoneum is not impaired, then the pathological process will be arrested here and recovery will follow. But should the effused blood be greater than the peritoneum can dispose of, even though there may be no visceral injury, decomposition will take place, probably through the agency of the colon-bacillus, the stagnant blood leading to the development of sapremia. Cases are not wanting to illustrate this; or local infection may lead to peritonitis. In wounds of solid viscera, even though hemorrhage is arrested, it is therefore manifest that sapremia and peritonitis may follow. In such cases the effused blood may be encapsulated, and subsequently undergo decomposition and pus-formation; these patients may then recover by evacuation of the abscess. But the usual course is death in a short time from sepsis. Should an intestine be perforated, immediately there is an escape of infectious gases. Within a short time, twelve to twenty-four hours, liquid contents will be extravasated. The effect of this is to produce the phenomena of perforative peritonitis, such cases often dying from the virulence of infection before there are marked changes in the peritoneum. The varieties of peritonitis incident to gunshot as well as to other visceral lesions, are discussed in detail under the caption "Peritonitis."

General Symptoms.—The location of the wound, the probable course of the bullet, its velocity as judged by the character of the weapon used, are questions to be taken into consideration in determining the probability of abdominal penetration. The re-

moteness of the wound of entrance from the abdominal area, as previously stated, does not eliminate the possibility of abdominal penetration. It is, therefore, all important that we consider the course and penetrating power of the missile. In any wound of the abdominal parietes penetration is to be presumed until disproved.

Here we must reiterate the statement that the general condition of a patient after intra-abdominal traumatism is influenced by the circumstanecs under which the injury is received, by the excitability or susceptibility of the individual to nervous impressions, by the nature of the vulnerating body, by the organ that is injured, by the physiological state of that organ, by the complicating injuries, and lastly and chiefly, by the occurrence of hemorrhage. These elements individually, in part, or collectively, contribute to the production of the physical phenomenon that we term shock.

Shock: Shock is the earliest and most constant of all symptoms, local or constitutional, of gunshot wounds of the abdomen. Nevertheless, it is so variable in degree that it is not surprising that in perhaps half of the cases reported, shock, when referred to, is described as slight or absent. The value of this symptom is greatly lessened by the knowledge that shock, even to a profound degree, may occur in non-penetrating wounds of the abdomen. Yet it can be maintained that in penetration of the abdomen such an impression is made upon the system in every instance that, if the patient is seen in time, that is, before reaction, the careful observer cannot fail to note some one of its phenomena. It may be expressed merely by pallor of the countenance, coolness of the body-surface, moist brow, or mental anxiety; or the patient may show deep collapse. Persistent shock invariably means hemorrhage.

Vomiting: Vomiting usually attends every penetrating wound. It does not necessarily denote visceral injury; it may be due entirely to the shock. Hematemesis may occur without gastric perforation, yet copious vomiting of blood would indicate a stomach-wound.

Melena: In intestinal perforation the effect of the injury is to arrest peristalsis; therefore, even if the bowel is wounded, we

are not likely to have bloody stools early enough to be of diagnostic import.

Hematuria: This, as indicative of renal, ureteral, or vesical injury, is more reliable than melena as affirmative evidence, but its absence does not preclude injury of these parts.

Pulse and **Temperature:** In 1890 Senn thus wrote: "The pulse at first is slow and compressible in all cases of gunshot wounds of the abdomen." The statement has been accepted as authoritative and unquestioning credence given to it, yet I must presume to say that it is too general and dogmatic. The pulse may be undisturbed. In profound shock without hemorrhage it may be abnormally slow and compressible. In slight shock, as gauged by all other symptoms, the pulse may be fast. The pulse does not immediately indicate the amount of visceral destruction. I have seen it full, strong, rating 90, with a portion of the bowel torn from its mesentery and protruding from the body. The value of the pulse is that of an indicator of the continuance of the hemorrhage. I have never seen a slow pulse when there was much bleeding. Only a few weeks before these lines were penned, Case XXI was seen one hour after the receipt of the wound; his pulse was 160 and barely perceptible. This is as near "at first" as patients can generally be seen. At operation one and a half hours later his abdomen was found to be full of blood. A shabby, wavering pulse indicates hemorrhage.

The central temperature depends upon the degree of shock. If there is marked general depression the temperature will be correspondingly low. After twelve hours both pulse and temperature become symptoms, not of penetration or perforation, but of the complications arising therefrom.

Pain: Pain is an indefinite, unreliable symptom. In wounds of the solid viscera, especially of the liver, it may not be noticeable. In wounds of the distal colon it is not marked. If the small intestine is involved it is usually griping, colicky in character, and as Miles, and Parker, of New Orleans, have remarked, is usually referred to the umbilical region.

If to these general symptoms there is added restlessness, thirst, anemia with syncope, dilated pupils, and cold extremities, the explanation will be found in internal hemorrhage.

PHYSICAL SIGNS.—The only positive sign of penetration is the presentation of the peritoneal surface of some viscus in the parietal wound. The escape of the contents of some one of the hollow viscera from the parietal wound is positive evidence of perforation, but not necessarily of penetration, since some of the viscera, notably the urinary bladder and colon, can be injured extra-peritoneally.

Hemorrhage: The surface wounds, of entrance or exit, rarely bleed freely. The abdomen may be distended with blood, but unless the wound be large and the track open, external hemorrhage will not appear. Free external hemorrhage is more likely to be due to wounds of parietal vessels.

Emphysema: Emphysema, so often referred to as a sign of visceral injury, is rarely seen except in wounds of the colon; a subperitoneal cellular emphysema then quickly develops. Oblique wounds may permit the entrance of air, and a crackling sensation may be detected by palpation.

Probing: The primary examination of the wound must be limited to inspection alone. Digital exploration is frequently impossible, often uncertain, and increases the danger of infection. The probe can rarely be conducted along the track of the wound, will follow without resistance the planes of the muscles, will often lead to the erroneous conclusion of non-penetration, and invariably introduces additional danger of infection. It should never be employed in the diagnosis of abdominal wounds except under the most rigid asepsis and preferably after the patient has been placed on the operating table, prepared for abdominal section.

Muscular Rigidity: Muscular rigidity is a valuable diagnostic sign of intestinal perforation. It is not so constant when the solid viscera alone are injured. It may be absent when shock is profound, or when hemorrhage is copious. It is easily detected in muscular and thin individuals; in obese patients it may escape notice. I esteem it of the highest diagnostic value.

Tympany: The sudden development of tympany, especially effacement of normal hepatic and splenic dulness, is a much vaunted sign of intestinal perforation. When it does suddenly appear within the first few hours after injury, then no doubt it is valuable; yet in only one of my twenty-one cases has it been present earlier than twelve hours. Its absence, then, as an early symptom, and the prob-

ability of mistaking a distended stomach or colon for free peritoneal gas, negative its value as a diagnostic sign.

Intraperitoneal Fluid: The detection of localized areas of abdominal dulness, suddenly appearing and progressively increasing, or the recognition of free intraperitoneal fluid soon after the infliction of the wound, could only mean hemorrhage, which must proceed from some serious intraperitoneal lesion.

Hydrogen-Gas Test: This test is highly satisfactory in an experimental way, and it reflects the greatest credit upon Professor Senn, yet it does not enjoy the approval of surgeons. That it is harmful in some instances we all believe. That it is uncertain, Dalton discovered to his misfortune, and Senn himself admits its unreliability. That it is only occasionally available every one must appreciate. I have not used it; I have never felt that a case permitted of the consumption of such valuable time as its employment would require.

SUMMARY OF SYMPTOMS: Guiding and specific information is not to be obtained from the semeiology of gunshot wounds of the abdomen. Shock is an intangible, indefinite symptom-compound, in some instances so slight as to be disregarded, and frequently marked when the injury is superficial and insignificant; its value must rest upon its persistency, its correlation with other symptoms. The pulse in its behavior is subservient to the shock. A rapid compressible pulse or a pulse varying in quality signifies hemorrhage; other than this it is not to be relied upon. Temperature is of no practical value in the first twelve hours. The pain, if about the umbilical region and colicky and griping in character, is of some significance, but like all subjective symptoms is uncertain. Neither vomiting, nor vomiting of blood, nor the passage of bloody stools or bloody urine, is absolute evidence of visceral perforation. The board-like abdomen—muscular rigidity—when present, is indicative of intestinal lesion, but we have shown that it may not be present in injury of the solid viscera, and in severe shock it may be absent; in obese subjects it is difficult to detect. Tympanites does not occur sufficiently early or often to be taken into consideraton for a timely diagnosis. Hemorrhage from the wound and emphysema about it are both frequently absent, and when present are not conclusive signs. The escape of visceral contents is an unusual phenomenon; in the pathology we

have attempted to show that from the stomach, urinary and gall-bladders there will be free and immediate extravasation; but in intestinal perforations early extravasation is an exceptional occurrence. In any event should extravasation occur, the size of the wound and its oblique course do not favor the appearance of visceral contents at the surface wound. In large wounds permitting free extravasation, the diagnosis of penetration with perforation is obvious.

It may be emphatically stated that the symptoms and signs as usually presented in gunshot wounds of the abdomen are not conclusively diagnostic of either penetration or perforation; that they do not with any degree of accuracy portray the certainty or seriousness of the visceral injury; and that from them we can not determine a prognosis. The mere knowledge of a wound of the abdominal parietes signifies nothing. The question to be determined, and upon the solution of which all others depend, is whether or not the missile has penetrated the abdomen. Confronted by the uncertainty of symptoms, and mystified by speculative opinion, we are eager to apply some positive, certain method of settling this all-important question. The hydrogen-gas test will not do, as we have shown. Probing and digital exploration are both fallacious and harmful. Therefore, in every case of wound that is not obviously penetrating, barring those cases of wounds in the back and those remotely located, the wound of entrance should be enlarged under cocain anesthesia, hemorrhage controlled by forceps-pressure, and by careful dissection by sight and with a grooved director its track should be followed until its penetrating or non-penetrating character is determined. If the ball has entered the peritoneal cavity, a surgical diagnosis is now made; and the exact injury that it has wrought within can only be revealed by laparotomy, the proper and imperatively demanded treatment. When the wound of entrance is not situated on the anterior or lateral aspect of the abdomen, when, in other words, the enlargement of the wound of entrance is not expedient for any reason, and the question of probable penetration presents itself; in such a predicament we must inquire minutely as to the line of flight of the bullet and its probable penetrating power, and attaching such significance to the complexus of symptoms presented as our judgment warrants.

If after careful investigation penetration cannot be excluded, abdominal section should be made.

PROGNOSIS.—Instances of recovery after known penetration without perforation have been referred to. Autopsies have also revealed the fact that death may follow simple penetration without perforation, such patients usually dying of sapremia. The gravity of any case depends largely upon whether or not there is visceral perforation; and it must be understood that a small, single, and apparently insignificant perforation may cause death. Those wounds are most serious which give rise to the greatest number and variety of injuries. Generally speaking, wounds of the solid viscera are not as fatal as those of the alimentary canal.

If the course of the bullet is anteroposterior the solid viscera alone may be injured; if intestines or stomach are perforated the number of such perforations is limited. If the direction of the ball is from side to side and above the umbilicus, we are likely to have wounds of solid viscera, intestinal perforation, and severing of large vascular trunks. For this reason I dissent from the statement, which is often made, that wounds above the umbilicus are less serious than those below it. It is true, those below the umbilicus pursuing a transverse or oblique course will almost certainly lead to multiple perforations; but it must be borne in mind that the immediate danger in wounds of the abdomen is hemorrhage, and low wounds are not so likely to sever large vascular trunks; and intestinal wounds do not bleed as freely as those of the kidney, liver, or spleen. Wounds from balls that enter in the rear, in addition to the difficulty of diagnosis, unless they pass entirely through the body, present additional dangers from hemorrhage, and because the perforations they produce offer difficulties both in detection and treatment. Wounds that involve the viscera of both abdomen and thorax are usually fatal in their termination.

It is only reasonable to anticipate greater injury in the track of a large bullet than in that of a small one; but the size of the ball is only one of several factors that influence the size of the perforation. It, therefore, has no reliable prognostic bearing. The cases of abdominal wounds at the battle of Santiago are too few for conclusions to be drawn, yet a report of them has somewhat advanced the cause of the conservatives. Parker says "there were about thirty such cases with a mortality of about 50 per cent." He attributes the recoveries of the patients to the small size of the bullet, the lack of food, and the prevalence of diar-

rhea. It is inferred from this that he thinks intestinal perforation occurred and spontaneously healed. Would not the empty, flaccid state of the bowel offer the greatest security against perforation? The evidence does not controvert the opinion that these abdominal wounds were instances of penetration without perforation, except, perhaps, in the single instance of Maj. Knox; and he had a wound involving the kidney and liver, and not the intestines. In no instance should the size of the bullet influence the treatment unless it be in wounds from a very small ball known to involve the liver alone, and in which no symptoms of hemorrhage develop.

In Siegel's table, compiled in 1898, of 532 wounds of the abdomen treated without operation, the mortality was 55.2 per cent. In 736 cases subjected to operation the mortality was 51.6 per cent. This showing taken without argument is to the advantage of operative intervention; and when we consider that there were at least one-third more cases operated upon than treated expectantly, that those that were subjected to operation were in all probability cases in which visceral injury was diagnosed or strongly suspected, and that in all penetration was proved, while in those not operated upon penetration may not have occurred, and at least 3 per cent. of them may have escaped perforation, it is, therefore, manifest that the good results of laparotomy in these 736 cases of gunshot wounds are better than the mere percentage of recoveries would indicate. A further analysis of this table shows that of those individuals operated upon within the first four hours the mortality was 15.2 per cent.; of those after twelve hours, the operative mortality was 70 per cent. In Fenner's series of 105 cases with visceral perforation subjected to operation 73.95 per cent. died.

In my table of cases, none of which is included in Siegel's list, and which are simply cases collected without selection from the literature since 1895, we have 65 cases operated upon, with 44 recoveries and 21 deaths, a mortality of 32.3 per cent.

Though the cases in my table are limited in number, it shows a mortality of only 32.3 per cent., while Siegel's shows a mortality of 51.6 per cent. This great difference can not be attributed alone to the difference in skill of the operators, for they are indiscriminately chosen. It is to be ascribed rather to the

recognition by the profession at large that penetrating wounds of the abdomen demand immediate surgical aid, and that the element of the time of operation most materially affects the result. Improvements in technic within the last five years have contributed somewhat to the good results, but the general employment of intravenous injections of saline solution in my individual experience has been of the greatest aid.

CONTRAINDICATIONS TO OPERATION.—Shock deepening into profound collapse, in which death seems imminent, contraindicates any surgical undertaking. If the patient is conscious, pupils not dilated, even though he may be pulseless, operation is not contraindicated. The surroundings of the patient may be such as to preclude the possibility of a successful abdominal section for injuries the exact extent and nature of which are unknown. Sufficient assistance may not be obtainable. Inexperienced help mars the technic, procrastinates the work, and materially affects the result. If the patient is doing well, and eighteen to twenty-four hours have elapsed, operation should not be done. If symptoms of peritonitis have developed there is small hope of recovery from operation. The necessity for extensive handling of inflamed peritoneal surfaces in the search for perforations makes surgery practically hopeless in these cases. There are few recoveries from operation done after twelve hours. In military surgery the relative infrequency of perforation following penetration, the fact that by reason of the emptiness of the hollow viscera and the small wound made by the modern military rifle bullet extravastion of the contents of the hollow viscera does not necessarily follow perforation, and the fact that there is frequently a delay of more than twelve hours before the patient can be gotten under surroundings that justify surgical intervention, makes this class of work to a great extent an exception to the rules that have been laid down.

GENERAL SURGICAL INDICATIONS.—**First Aid**: In civil practice the most important feature of first aid in gunshot wounds of the abdomen is to refrain from meddlesome handling of the patient and his wound. It is probably best under all circumstances to give a dose of morphia hypodermically. This checks any tendency to peristalsis on the part of possibly injured bowel, quiets the patient's nerves, relieves pain and also shock to some extent. The morphia

should be repeated, if needed, for pain. Stimulants are of doubtful value, and if used at all should only be given hypodermically. The patient's wound should be left alone. In most instances it is best not even to unfasten the patient's clothing. There is no hemorrhage that can be controlled until the abdomen is opened; in fact, there is nothing that can be done for the injury itself until the patient has been placed under the proper surroundings for surgical intervention.

Operation: After thorough investigation of the case, according to the plan already outlined, if it is found that abdominal section is necessary, it should be made at once. Ether is preferred as the anæsthetic. The incision should be made in the median line unless the known course of the bullet justifies a lateral opening. Certainly a median incision should be employed, whenever the wound of entrance or exit lies near the median line, and whenever the bullet crosses a median plane. The incision wherever placed should be made of ample length.

Once within the abdomen, the first step is to control hemorrhage. This is usually venous or parenchymatous, and can ordinarily be stopped by pressure. If the area from which the hemorrhage comes can be located pressure should be made upon it with gauze pads. Active stimulation with normal salt solution and suprarenal extract may now be used if the patient's condition demands it. It is hardly rational to use it earlier. With the bleeding temporarily held in check by pressure the abdomen is emptied of blood by sponging. Once this is accomplished, the pads are carefully shifted to uncover the bleeding points and permanent hemostasis established by ligature, suture, tampon or forci-pressure. The actual cautery should never be used except as a last resort.

Any intestinal perforations found during the search for the source of hemorrhage should be closed by immediate suture, if small, or by rubber-covered clamps, if large enough to require much time or to raise the question of resection. However, when the hemorrhage is profuse, it is probably best to ignore all other considerations until it is under at least temporary control.

The next step is to repair visceral injuries by methods which will be taken up under separate heading.

The peritoneal cavity should be sponded free of extravasated material or lavage may be used if necessary for this purpose.

Drainage should only be used when there are areas of badly damaged tissue in which we fear necrosis coming up later, or where tamponage is necessary to control hemorrhage.

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GUNSHOT WOUNDS OF THE STOMACH.

FREQUENCY.—The great expanse of the stomach-wall when the organ is distended, its close relation to the parietes, and its fixed position, render it liable to penetration. After wounds of the intestines and liver, those of the stomach come next in order of frequency. It is true, only 64 cases came under treatment during our Civil War; but, as Col. Otis remarks, the greater proportion of injuries of this class must be sought for in those killed in action.

PATHOLOGY.—We find upon investigation that wounds of the stomach are usually complicated in their nature. The pancreas, liver, transverse colon, spleen, and left kidney are the abdominal organs conjointly injured. The wound of entrance is frequently through the thorax, so that we also have injuries of the thoracic viscera and diaphragm, a complication usually fatal in its nature, and one that often follows awkward attempts at suicide. In complicated injuries there is usually profuse hemorrhage. This is of diagnostic significance, as simple wounds of the stomach from missiles of moderate size do not bleed copiously, unless, perchance, a large vessel has been severed.

The condition of the stomach at the time of injury materially influences the size of the perforation. A ball entering a distended stomach will not only produce a larger opening, but the lateral force will be evidenced by ecchymoses, if not by actual lacerations, beneath the peritoneal covering of the stomach. I make this statement based upon one observation only. In one of my cases I found two perforations of the anterior wall of the stomach. I noticed beneath the serosa of the stomach-wall, remote from the track of the bullet, two or three ecchymotic spots. Their isolated position could only be explained by the explosive effect of the bullet.

Extravasation occurs more frequently and earlier in stomach-wounds than in intestinal injuries; vomiting causes the extrusion of the contents.

Perforations made by balls of small size, .32 or less, are not easily discoverable. The discoloration beneath the serosa surrounding the perforation marks the site of injury and is the best guide for its detection. I have not seen ectropion of the mucous membrane in stomach-wounds.

The gastric contents are chemically irritating, but not as virulently septic as the contents of the intestines; therefore, in small wounds, in which the extravasation is limited in quantity, there is a probability of the formation of a circumscribing inflammation, and the result may be an abscess with gastric fistula. Such a favorable course after extravasation is not to be expected. Infection with local gangrene or peritonitis develops, which may at first be limited to the upper zone of the abdomen but soon becomes general. Wounds of the posterior wall of the stomach may not prove immediately fatal, but the leakage into the lesser peritoneal cavity and infection give rise to subphrenic abscess.

Spontaneous healing without fistula may take place; this can only occur when the perforation is small and when no extravasation has occurred; Manley records one case of his own, and three others, in which the evidence seems indubitable. An added seriousness is given to cases of gunshot wound of the stomach by the pancreatic injury which so frequently complicates them.

Symptoms.—In gunshot wounds of the stomach we have the general symptoms common to wounds of the abdominal cavity, shock, pain, muscular rigidity, etc. The symptom peculiar and diagnostic of stomach-lesion is the vomiting of blood. This may occur without perforation of all the coats of the stomach, but clinically the vomiting of blood, either free or mixed with stomach-contents, should be accepted as evidence of gastric or duodenal perforation. Persistent vomiting, localized pain, hiccough, and pronounced nervous disturbances are usually prominent symptoms in gastric wounds. If the perforation is large, permitting extravasation, tympany rapidly develops, and the symptoms of general peritonitis follow. Inasmuch as wounds of the stomach are generally complicated, shock of a profound degree, as a rule, characterizes these injuries; it is due either to the multiplicity and severity of the complications, or to hemorrhage. In combined thoracic and abdominal wounds the respiratory distress may overshadow the abdominal symptoms.

The hydrogen-gas test should not be used in wounds of the stomach. The passing of the tube excites additional vomiting and greater straining, and severely taxes an already weakened patient. The distention of the stomach would certainly favor extravasation. If the diagnosis of gastric perforation is doubtful the question should be settled by the general law that should obtain in all penetrating wounds of the abdomen: Expose by celiotomy the abdominal viscera for visual examination, and search for perforations.

PROGNOSIS.—Since the days of Alexis St. Martin the profession has been impressed that wounds of the stomach behave more kindly than those of other portions of the alimentary canal, and the prevailing tendency, therefore, is to treat them conservatively. Laplace says that small stab- and gunshot wounds of the stomach yield without trouble. Manley says that they should not be interfered with unless there is great extravasation. I must dissent from this favorable prognosis. The general mortality is placed at 90 per cent. MacCormac says that 99 per cent. of gastric perforations terminate fatally. Otis found only one incontestable case of recovery out of 64 perforations of the stomach. In the appended table of gunshot wounds of the stomach there are ten cases subjected to operation, with seven recoveries and three deaths.

SURGICAL INDICATIONS.—Wounds of the stomach if small should be closed by purse-string or other seromuscular suture. If large, two layers of suture should be put in, one including all the coats of the organ and the other seromuscular. Wounds of the posterior wall can usually be reached most satisfactorily by opening the lesser cavity of the peritoneum through the gastrocolic omentum. Occasionally it will be necessary to make an anterior gastrotomy to reach injuries of the posterior wall. When drainage of the lesser cavity is required, it will usually be by reason of complicating injury to the pancreas, and should be put in through the lumbar region if possible.

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GUNSHOT WOUNDS OF THE INTESTINES.

FREQUENCY.—Relatively speaking, the intestines are wounded more frequently than any of the abdominal viscera. Their size, length, position, and lack of bony protection explain this. A bullet with an anteroposterior course may in exceptional instances pass between the coils of the intestines, or may perforate the solid viscera, the intestines escaping; but in all penetrating wounds in which the bullet pursues an oblique or transverse course through the abdomen, multiple perforation is the rule; the exceptionally harmless behavior is purely accidental and does not influence the general law. The statement is made that in penetrating wounds of the abdomen the intestines are perforated in 65 per cent. of cases. In my table of collected cases I find that in 68.6 per cent. of cases there were uncomplicated wounds of the intestines.

PATHOLOGY.—The immediate effect of a perforation on the normal intestine is to excite a spasm which results in ectropion of the mucous membrane. This acts as an obturator to the perforation, and prevents the escape of intestinal contents. Nature further subserves this purpose by arrested peristalsis and adhesive inflammation. In long wounds of the gut, severing for an inch or more the circular fibers, the central portion of the wound does not show the same protrusion as is evident at the extremities. When the intestine is severed entirely the mucous membrane rolls out of the free ends and gives the extremities almost a clubbed appearance.

Hemorrhage is not profuse when the wound involves the wall of the gut alone. The mucous membrane prevents the blood from entering the channel of the bowel, and hence in many instances of multiple perforation blood in noticeable quantity does not appear in the stools.

Extravasation in gross amount does not take place until the intestines yield to paralytic distention, or under operative manipulation. In Case V of my series, in which there were thirteen

perforations, the patient walked one mile after being shot, was not operated upon for twenty-two hours, and yet there was no noticeable extravasation.

Wounds of the mesenteric border or of the principal mesenteric arteries and veins, without perforation of the intestines, are especially dangerous because of the liability to gangrene of that portion of the intestines supplied.

Should the patient escape immediate death from hemorrhage, perforation-peritonitis with its violently septic and rapidly fatal course develops; it is only in exceptional instances that the patient withstands the attack longer than forty-eight hours. The effort to circumscribe the injured area is futile, and a general diffuse peritoneal inflammation results. Should the patient survive longer than twenty-four hours, suppurative peritonitis will be found at autopsy. In wounds of the large intestine, intra- or extraperitoneal, the possibility of recovery with fecal fistula is infinitely greater than in those involving the small intestine.

We can not deny the possibility of spontaneous healing of very small intestinal perforations; yet all instances of its reported occurrence are, to say the least, questionable. Lange's case is, perhaps, the most authentic, and even in it the perforation may not have been complete. If healing does occur it must be by agglutination of a contiguous serous surface. The great hindrance to healing is the eversion of the mucosa.

Symptoms.—Under the head of General Symptoms of Gunshot Wounds of the Abdomen the symptoms of intestinal perforation were considered. It is only necessary here to say that shock may be variable in degree, often so slight as to be unnoticed; that patients are not immediately stricken down by such an accident we have numerous examples to show. In one of my cases with thirteen perforations the patient walked a mile without assistance. Pallor and vomiting are two symptoms generally present. After them I would place muscular rigidity; and I must insist that it points strongly to acute intraperitoneal injury in general, and to intestinal perforation in particular. The pulse and temperature are not primarily of great value, except as denotive of profound shock from hemorrhage; later, they become of diagnostic and prognostic import. Shallow and frequent respiration is a symptom of abdominal distress. LeDentu regards a respi-

ration of twenty-eight or thirty an absolute indication of perforation. At first it may be an emotional symptom and, unassociated with other symptoms, has no such value. Hyperesthesia of the abdomen may occur in parietal wounds alone, but hyperesthesia associated with general rigidity is significant of internal injury. If extravasation of visceral contents occurs, which is exceedingly unlikely as encountered in civil practice, it must be accepted as conclusive evidence of visceral perforation; but, as Murphy says, it is folly to wait for extravasation to appear. Free bleeding from the surface wound may be from a parietal vessel. Unless the wound is large, direct, and over dependent areas, the intraperitoneal hemorrhage will not appear externally through it. The presence of free fluid in the abdominal cavity, or the detection of areas of abdominal dulness progressively enlarging, associated with the phenomena of shock, can only mean hemorrhage. Bryant has cautioned us against turning the patient, rude palpation and percussion, sometimes employed for the detection of free peritoneal fluid. In the general symptomatology we have indicated the objections to probing, digital examination of the wound, and Senn's hydrogen-gas test.

If the symptoms of penetration are not clear the diagnosis must be made by enlarging the wound of entrance, if this is possible, carefully dissecting along the track until its course is determined. If it is found to be penetrating it must be assumed, until otherwise proved by exploratory incision, that there is perforation of some of the viscera; and in 65 per cent. of all cases of penetrating wounds of the abdomen the intestines are perforated.

PROGNOSIS.—The mortality of gunshot wounds of the intestines is variously placed at from 80 per cent. to 90 per cent. As the question of intestinal perforation in all cases conservatively treated which recover will forever remain uncertain because of the want of positive signs of perforation, the true percentage of recoveries after such an injury can not be estimated. Proof seems to be accumulating that penetration without perforation occurs in perhaps a greater number of cases than we now estimate, 3 per cent.; but there is no proof offered that perforating intestinal wounds, no matter how small the bullet, how empty the alimentary canal, can be regarded in any other than the gravest possible light.

Of 45 cases subjected to operation 31 recovered, a recovery rate of 67 per cent. A review of these cases and the injuries found would convince one of their utter hopelessness without operation. I have personally within the last five years operated upon eight cases of gunshot wounds of the intestines, with a result of six recoveries and two deaths; all except one were operated upon within four hours from the time of injury. In one case in which there were ten perforations with entire severance of the ileum, death occurred on the sixth day from peritonitis due to leakage at the site of the Murphy button. The other patient died on the thirteenth day from peritonitis due to a mural abscess; the intestinal wounds had healed perfectly.

TREATMENT.—In order to locate intestinal injuries it will usually be necessary to search the whole length of small intestines, but the possible location of injuries to the colon can be more definitely determined beforehand, so that only a portion of that viscus will need to be investigated.

In looking over the small intestine the loop used as a starting point must be held by an assistant, and so held that the direction in which the search is first prosecuted can be told when the operator is ready to search in the opposite direction. Perforations that can be satisfactorily closed by suture should be sutured as found, but resection should never be done until the whole bowel has been investigated. The manipulation necessary to resection and anastomosis tends to provoke peristalsis and might lead to extravasation from uncontrolled openings. Then, too, another injury demanding resection might be close at hand, and a single resection, including the intervening bowel, might be preferable. For these reasons injuries requiring resection should be controlled by clamps until the search is completed.

Perforations on the free border of the small intestine, if quite small, may be closed by purse-string suture. Larger perforations should be closed in the short axis of the bowel by seromuscular sutures. Occasionally two layers of sutures, one through and through and one sero-muscular, will be required. If the wound is so large that suturing in this fashion tends to cause severe kinking, the line of closure should be placed in the long axis of the bowel, provided the lumen is not diminished more than one half by

this procedure. If closure by suture of an injury causes too much encroachment upon the lumen of bowel, resection is demanded.

Perforations at the mesenteric border of the small intestine will frequently demand resection on account of interference with the blood supply of the corresponding section of the bowel. The rule usually followed is that injuries in this situation involving as much as half an inch of the long axis of the gut demand resection. This is, of course, arbitrary and should not be followed too closely, but in case of doubt resection should be done. Wounds of the mesentery itself in the area of the vasa recta should be treated as wounds of the mesenteric border of the bowel.

Wounds of the small intestine between the mesenteric and free borders must be handled according to size and situation. Those nearer the mesenteric border are apt to cut off the blood supply of the corresponding portion of the free border, and may demand resection on this account when otherwise they could be closed by suture.

In closing perforations a wide margin of the bowel should be turned in to allow for necrosis, and this is especially necessary on the mesenteric border, the necrotic process here having a tendency to encircle the bowel. The larger injuries require two layers of sutures, one through all the coats, and another seromuscular.

When resection is required anastomosis should be made by the suture method. The Murphy button is only justified by inexperience on the part of the operator.

Perforations of the large intestine are closed just as are injuries of the small bowel, except that the direction of the line of sutures is of less importance. Resection is seldom required. When it is, anastomosis should be made if possible, otherwise the ends of the bowel must be closed and a communication established between the proximal and distal portions of the gut.

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GUNSHOT WOUNDS OF THE LIVER.

FREQUENCY.—Wounds of the liver occur in point of frequency next to those of the small intestine.

PATHOLOGY.—The immediate effect of perforation of the liver is hemorrhage. The size of the wound and its situation in the liver, of course, influence the amount of hemorrhage. Central wounds, severing large vascular trunks, either within the liver-substance or as they enter, may prove immediately fatal. Wounds that extend from the free border of the liver into its substance, leaving a gaping fissure, bleed more copiously than those that merely perforate its substance.

The escape of bile into the peritoneal cavity, which inevitably occurs in wounds of the liver, does not immediately precipitate a peritonitis. The innocuousness of pure bile to the peritoneum has been repeatedly demonstrated experimentally and proved clinically; yet the continued escape of a large quantity of bile ultimately produces death from cholemia or peritonitis. In Case IV of my series, free drainage was not obtained until the patient showed deep icterus, with the symptoms of which he died, there being no peritonitis.

In wounds of the liver, the immediate danger, hemorrhage, is within the first twenty-four hours. The remote or complicating pathological results may not terminate for several weeks. Wounds of the liver are usually complicated with wounds of the stomach, intestines, and kidney. More serious and frequent complications are wounds of the thoracic viscera, diaphragm, gall-bladder, and portal vein. In Case VII the wound of entrance was just below and to the right of the right nipple; in this case there were wounds of the pleura, diaphragm, liver, portal vein, transverse colon, small intestine, and sigmoid flexure. The wound of exit from the abdominal cavity was in the iliac fossa. The complications here presented are as serious as one could well conceive of.

SYMPTOMS.—In uncomplicated wounds of the liver, unless there be hemorrhage. the symptoms may be unimportant, and are

rarely diagnostic in character. In such instances we can only infer that the liver is injured by the location of the wound of entrance, and the direction of the bullet. Usually, however, in addition to these facts in the history, the patient presents symptoms of hemorrhage. Pain may be great or unnoticed; it is only significant when referred to the shoulder. Wounds of the liver and other solid viscera, as previously stated, do not cause marked muscular rigidity. In one-fifth of the cases Edler says icterus occurs. The primary icterus is due to the absorption of bile effused into the peritoneal cavity. Itching of the skin, nausea, and mental hebetude attend the development of cholemia. If the patient escapes septicemia and peritonitis the symptoms may be very different from those of any other character of abdominal wound, pursuing the course of acute cholemia. This was strikingly illustrated in Case IV. In wounds of the gall-bladder or duct the escape of bile is so rapid that death usually occurs very quickly.

The complications that attend wounds of the liver are generally of greater importance symptomatically and surgically than the hepatic wounds themselves. In a recent case of mine the wound of the liver did not require treatment, but the complicating wound of the kidney presented urgent symptoms.

Prognosis.—In "The History of the Rebellion," of the 59 uncomplicated wounds of the liver recorded 25 recovered. Edler places the mortality at about 40 per cent. The mortality of complicated wounds depends entirely upon the nature of the complication. The table at the close of this article shows 8 cases: 6 were operated upon, with 4 recoveries and 2 deaths; 2 treated expectantly, both died. In Finkelstein's series of 13 cases of hepatic wounds operated upon 8 recovered.

Treatment.—Uncomplicated wounds of the liver frequently do not require surgical intervention, but the impossibility of excluding complications makes exploration essential. In punctured wounds hemorrhage can usually be controlled by packing. Cotton that has been wet and wrung dry is more efficient than gauze as a tampon for controlling hemorrhage. When there has been a fracture of the liver or a groove cut into it, sutures may be required. For this purpose a straight blunt needle and large catgut is to be preferred.

All kinks should be carefully stretched out of the catgut before it is drawn through the needle wounds in the liver.

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FINKELSTEIN. Roussky Vratch, 1902.

GUNSHOT WOUNDS OF THE PANCREAS.

Wounds of the pancreas are very likely to escape detection, even at operation. They are always complicated with injuries of other viscera, and sometimes large vascular trunks are severed, patients usually dying of such a wound before surgical relief can be afforded. Otis records five cases of wounds of the pancreas during the Civil War. In one of the five there was hernia of the pancreas, and a portion of it was removed; this case recovered. In the remaining four the wound of entrance was in the rear. Three of the cases lived from twelve to fifteen days. The treatment of gunshot wounds of the pancreas includes the control of hemorrhage by suture and tampon, and the establishment of ample tube and gauze drainage. Lumbar drainage should be used whenever possible, even if anterior drainage is also required.

GUNSHOT WOUNDS OF THE SPLEEN.

FREQUENCY.—In review of the cases reported, we find comparative infrequency of gunshot wounds of this organ; it is far more liable to injury from contusions.

PATHOLOGY.—The friable nature and extreme vascularity account for the free bleeding which occurs in wounds of the spleen. Wounds of the spleen are frequently complicated by wounds of the left kidney, stomach, diaphragm and intestines. Case VI of Fenner's personal series in which there were wounds of thorax, diaphragm, stomach, spleen and liver, illustrates the complications, and the recovery of the patient after splenectomy demonstrates the possibilities of prompt surgery. If the patient survives the immediate hemorrhage, the subsequent intraperitoneal pathological process is the result of decomposition and absorption of ptomains, or of infection from other visceral injuries.

SYMPTOMS.—Wounds of the spleen are recognized by the location of the wound of entrance, the course of the missile, and by the symptoms, local and constitutional, of hemorrhage. The shock of anemia rapidly develops. The extravasated blood shows a tendency to accumulate at first in the left hypochondriac region, and the progressive extension of this area of dulness, with gradually failing pulse, increasing pallor, thirst, and restlessness, all indicate the continued hemorrhage. The constancy of complications in wounds of the spleen accounts for the complexus of symptoms presented.

PROGNOSIS.—The copious hemorrhage which attends wounds of the spleen, the difficulty met with in controlling it, and oftentimes the necessary splenectomy upon an exsanguinated patient, and the probable complications of wounds in the kidneys, intestines, or stomach, all contribute to make gunshot wounds of the spleen exceedingly dangerous.

SURGICAL INDICATIONS.—Wounds of the spleen give rise to such copious hemorrhage that relief must be quickly afforded to avail. The special indication is to control hemorrhage. Tiffany has

shown us that this may sometimes be successfully done with suture. Should this effort be unsuccessful, *splenectomy* is the only alternative—a difficult procedure with a spleen of normal size.

GUNSHOT WOUNDS OF THE KIDNEY.

FREQUENCY.—Gunshot wounds of the kidney rarely occur without complicating wounds of the liver, spleen, or intestines; and we find it difficult to collect a sufficient number of independent kidney-wounds to be of any material value.

PATHOLOGY.—Wounds of the kidney may involve the peritoneum or be entirely external to that membrane. If the peritoneum is penetrated, complicating wounds of other viscera are the rule; wounds of the colon, liver, and spleen are the most frequent associate injuries. The chief pathology, after hemorrhage, is due to the extravasation of urine. If the peritoneal surface of the kidney is wounded the escaping blood and urine is intraperitoneal; it meets with less resistance in that direction than into the perirenal tissues. There is great liability to infection and suppuration in kidney-wounds. Wounds of the convex border and extremities of the kidney are much less serious than those of the central portion. If the ureter is severed or calices opened, there is free escape of urine, and usually profuse hemorrhage.

SYMPTOMS.—The symptoms especially significant of renal perforation are pain and hematuria. The immediate discharge of urine through the external wound rarely occurs as a primary symptom. The pain is somewhat diagnostic in character; it radiates downward; it is felt in the genitals and thigh; and with it there is usually retraction of the testicle and great desire to urinate. Hematuria, as we have previously shown, may occur without an actual wound of the kidney. Clinically, however, we attach the greatest value to this symptom. It is generally present in wounds of any portion of the kidney. It may be absent from a variety of causes; if the ureter is severed the few drops of blood that find their way into the bladder may escape detection; if blood accumulates in the pelvis of the kidney it will occlude the ureter, and only clear urine from the other kidney will reach the bladder; if the wound is extensive and hemor-

rhage great, the loss of vascular tension may suspend the secretion of urine from that kidney, and there will be no blood in the urine. The hemorrhage from wounds of the kidney is frequently intermittent. In Case XIX of my series the patient had urinated about every hour for the first twelve hours; his desire grew less urgent, and at intervals of three hours he passed some four ounces of clear urine. When I reached him, about twenty hours after the accident, he had just voided urine containing a quantity of blood. The passage of clots of blood along the ureter excites spasms of pain, and attending these there is usually desire to void urine. The rapid development of a loin tumor, as indicated by abnormal dulness and by palpation, suggests the possibility of renal injury with hemorrhage.

The other symptoms of renal perforation are common to perforating wounds of the abdomen in general. Their gravity depends upon the amount of hemorrhage and upon associate complications.

Prognosis.—Edler places the mortality of uncomplicated wounds of the kidney at 44 per cent.; the general mortality of all kidney-wounds at 47.3 per cent., less than in any other of the fixed abdominal viscera. Of 38 bullet-wounds of the kidney tabulated by Tuffier, 22 recovered and 16 died.

Surgical Indications.—As wounds of the kidney are usually complicated with other visceral injuries, abdominal section is generally necessary. The special management of the kidney-wound depends upon the extent and location. If the bullet has severed the renal vessel or wrought great destruction of kidney tissue, nephrectomy is demanded. Wounds of the pelvis or ureter require nephrotomy for temporary drainage of the urine. Hemorrhage is controlled by suture or tampon and ample drainage through a lumbar incision should always be provided.

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Tuffier. Archiv. gén. de Méd., March, 1889.

GUNSHOT WOUNDS OF THE STOMACH, 1895–1900.

SEX. AGE.	LESIONS FOUND.	TREATMENT.	RESULT.	AUTHOR.	REFERENCE.
1. M. 28.	Perfor. of stomach, and 7 perfors. of small intestine.	Op. 2 hrs. p. tr. Shock.	Death 15 hours p. op.	C. A. Atwood.	Bost. Med. and Surg. Jour., 1900, CLXII.
2. M. —	Perfor. of stomach near cardia.	Op. Shock.	Death 46 hrs. p. op.	W. K. Sheddan.	Nash. Jour. Med. and Surg., 1899, LXXXVI.
3. M. Adult.	5 perfors. of stomach and 3 of small intestine.	Op. 7½ hrs. p. tr.	Recovery.	C. E. Miller.	Amer. Jour. Surg. and Gyn., 1899, XIII.
4. F. 16.	Perfor. of stomach.	Op. 2 hrs. p. tr. Shock.	Recovery 1 month.	J. W. Cousins.	Br. Med. Jour., 1898, II, 145.
5. M. 13.	Perfor. of stomach and small intestine.	Op. 6 hrs. p. tr.	Recovery 16 days.	A. M. Vance.	Amer. Prac. and News, Dec., 1898.
6. M. 44.	Perfor. of stomach and duodenum.	Op. 1 hr. p. tr.	Recovery 15 days.	"	Lon. Jour. Surg. and Med., March, 1899
7. F. 30.	Perfor. anter. wall of stomach.	Op. 3 hrs. p. tr.	Recovery 21 days.	A. H. Garratt.	Can. Med. Rev., April, 1898.
8. M. 58.	Perfor. anter. wall of stomach.	Op. soon p. tr.	Recovery 1 month.	R. Winslow.	Annals of Surg., 1898.
9. M. 14.	Perfor. of stomach. Autopsy: Broncho-pneumonia; perfect union of perforation.	Op. Shock.	Death 5 days p. tr.	T. H. Openshaw.	Trans. Path. Soc., London, 1895, XLV.
10. M. 19.	Perfor. of stomach and mesentery.	Op. ¼ hr. p. tr. Shock.	Recovery 1 month.	G. S. Reanie.	Internat. Jour. Surg., 1895, III.

Gunshot Wounds of the Intestines, 1895–1901.

Sex. Age.	Lesions Found.	Treatment.	Result.	Author.	Reference.
1. M. Adult.	4 perfors. of small intestine.	Op. 14 hrs. Shock.	Death 12 hrs. p. op.	Auvray and Rochard.	Bull. et Mém. d. l. Soc. Chir., Paris, 1901, XXVII.
2. F. Child.	15 perfors. of intestines.	Op. Shock.	Recovery.	Payton Beale.	Med. Press and Cir., Lon., 1900, LXX.
3. M. 24.		No op. No shock.	Recovery.	J. R. Hicks.	Va. M. Semi-Month., 1900, V.
4. M. 20.	4 perfors. of jejunum, 2 of mesentery.	Op. 1 hr. No shock.	Recovery.	A. P. Gould.	Tr. Clin. Soc., Lon., 1900, XXXII.
5. M. —	3 perfors. of ileum.	Op. 14 hrs. p. tr.	Recovery.	H. H. Grant.	Ga. J. M. and S., 1900, VI.
6. M. 9.	2 perfors. of small intestine and mesentery.	Op. 22 hrs. Shock.	Recovery in 50 days.	E. Pernitzer.	Wien. klin. Woch., 1899, XII.
7. M. Adult.	2 perfors. of small intestine.	Op. several hrs.	Death 12 hrs. p. op.	Haga.	C. R. de XII Cong. Internat. de Méd., 1899, V, III.
8. F. 29.	2 perfors. of small intestine.	Op. immed. No shock.	Recovery in 27 days.	J. Frank.	Med. Rev., St. L., 1899, XL.
9. M. 26.	2 perfors. of small intestine and mesentery.	Op. immed. Shock.	Recovery in 60 days.	R. E. Fort.	Nash. J. M. and S., 1899, LXXXVI.
10. M. Adult.	16 perfors. of intestines and mesentery.	Op. ¾ hr. p. tr.	Death during op. due to ether.	Flagg.	Rep. Surg. Gen., 1899, 322.
11. M. Adult.	7 perfors. of small intestine.	Op. 10 hrs. Shock.	Recovery.	C. E. Miller.	A. J. Surg. and Gyn., 1899, XIII.
12. M. 34.	2 perfors in colon, 2 in loop of intestine behind colon.	Op. ½ hr. No shock.	Recovery in 46 days.	N. E. McKay.	Phil. M. J., 1899, I.
13. M. 60.	4 perfors. of small intestine.	Op. 18 hrs. No shock.	Recovery.	R. Winslow.	Annals Surg., 1898.
14. M. 25.	5 perfors. of ileum, 3 of mesentery.	Op. 2 hrs. No shock.	Death in 4 days.	"	"
15. M. 67.	7 perfors. of small intestine.	Op. 1 hr. No shock.	Death next day.	"	"
16. M. 44.	6 perfors. of small intestine, 1 of omentum.	Op. 2 hrs. No shock.	Recovery in 48 days.	"	"

Sex.	Age.	Lesions Found.	Treatment.	Result.	Author.	Reference.
17. M.	23.	1 perfor. of first portion of duodenum.	Op. 11 hrs. No shock.	Recovery.	R. Winslow.	Annals Surg., 1898.
18. M.	27.	5 perfors. of small intestine, 3 of mesentery.	Op. 2 hrs. Shock.	Death 3 days p. op.	"	"
19. M.	16.	6 perfors. of small intestine; mesenteric vein severed.	Op. 8 hrs.	Recovery in 6 wks.	R. W. Brown.	Va. M. Semi-month., 1898.
20. M.	—	6 perfors. of small intestine and mesentery.	Op. 1 hr. No shock.	Recovery.	C. R. Parke.	N. Y. Med. J., 1898.
21. M.	11.	2 perfors. of ascending colon.	Op. 24 hrs. No shock.	Recovery.	G. N. Josserand.	La Province Méd., Lyon, 1898, XIII.
22. M.	Adult.	12 perfors. of intestines.	Op. 4 hrs. Severe shock.	Recovery in 4 wks.	Poppert.	Arch. f. klin. Chir., 1898, LVIII.
23. M.	15.	6 perfors. of ileum, 1 of tr. colon.	Op. 2½ hrs.	Recovery in 6 wks.	Schnitzler.	Wien. klin. Rundsch., 1898, XII.
24. M.	—	12 perfors. of mesentery, artery severed.	Op. during shock (?).	Recovery in 4 wks.	W. F. Cole.	Med. Sentinel, Oregon, Oct., 1898.
25. M.	47.	2 perfors. of small intestine.	Op. in a few hrs. Severe shock.	Death in 10 hrs.	W. Gillette.	Med. Record, 1898.
26. M.	40.	7 perfors. of small intestine and tr. colon.	Op. 8 hrs. No shock.	Recovery.	"	"
27. M.	22.	2 perfors. of ileum, resection 20 ins.	Op. 2 hrs. p. tr.	Recovery.	H. H. Grant.	S. S. and Gyn. Trans., 1898, XI.
28. M.	28.	3 perfors. of ileum, 4 of mesentery.	Op. 4 hrs. p. tr.	Recovery.	"	S. S. and Gyn. Trans., 1898, XI.
29. F.	31.	5 ins. resected, 2 perfors. sutured.	Op. 2 hrs. p. tr.	Recovery.	"	S. S. and Gyn. Trans., 1898, XI.
30. M.	30.	2 perfors. of mesentery, 5 ins. intestine resected.	Op. 2 hrs. p. tr.	Death in 30 hrs. from peritonitis.	"	S. S. and Gyn. Trans., 1898, XI.
31. M.	—	Evidently no perforation.	No op. No shock.	Recovery in few days.	Harvey Reed.	J. A. M. A., June 4, 1898.
32. M.	15.	2 perfors. of jejunum, 1 of ileum.	No op. No shock.	Recovery in 3 wks.	J. L. Wiggins.	Med. Rev., St. L., June 18, 1898.

GUNSHOT WOUNDS OF THE INTESTINES, 1895–1901. (*Continued.*)

SEX. AGE.	LESIONS FOUND.	TREATMENT.	RESULT.	AUTHOR.	REFERENCE.
33. M. 14.	Perfor. of jejunum. Autopsy: perfors. of abdom. aorta, stomach and gall bladder.	Op. 3 hrs. p. tr.	Death in 19 hrs. from hemorrhage.	C. C. Booth.	Cleveland Med. Gaz., 1897, XII.
34. M. 20.	Wound of small mesenteric vein.	Op. during shock.	Recovery.	W. W. Mayo.	Br. Med. J., July 10, 1897.
35. M. 35.	Perfor. of ileum, 2 of mesentery, 2 of descending colon.	Op. at once.	Recovery in 4 wks.	O. Suter.	Med. Rev., St. L., 1896, XXXIII.
36. M. 11.	12–15 perfors. of small intestine.	Op. 6 hrs. No shock.	Recovery in 22 days.	J. Donnelly.	Toledo M. and S. Rep., 1896, IX.
37. M. 36.	Perfor. of descending colon. Next day, hematuria,—probably rupture of kidney. No autopsy.	Op. 2 hrs.	Death next day.	"	Toledo M. and S. Rep., 1896, IX.
38. M. 17.	5 perfors. of intestine, 1 of bladder.	Op. at once. Shock.	Death 24 hrs. p. tr.	Fred. Page.	Br. Med. J., 1896, II.
39. M. Adult.	2 lacerations of small intestine, great omentum and liver.	Op. Shock.	Death 12 hrs. p. op.	D. M. Appel.	Surg. Gen. Rep., 1895.
40. F. —	3 perfors. of small intestine, 1 of colon.	Op. 4 hrs. p. tr.	Recovery.	Richard Douglas.	——— 1897.
41. M. 21.	4 perfors. of small intestine.	Op. 3 hrs. p. tr.	Death in 13 days.	"	J. A. M. A., March 18, 1899.
42. M. 17.	7 perfors. of omentum, 2 of colon.	Op. 2 hrs. p. tr.	Recovery.	"	J. A. M. A., March 18, 1899.
43. M. 20.	4 perfors. of intestine.	Op. 4 hrs. p. tr.	Recovery.	"	J. A. M. A., March 18, 1899.
44. M. 29.	6 perfors. of intestine, 1 of small mesenteric artery.	Op. 2 hrs. p. tr.	Recovery.	"	J. A. M. A., March 18, 1899.
45. F. 15.	Laceration of 5 ins. of ileum, severed and torn from mesentery, protruding through large parietal wound.	Op. 3 hrs.	Recovery.	"	J. A. M. A., March 18, 1899.
46. F. 20.	Perfor. of descending colon.	Op. 3d day.	Recovery.	"	——— 1899.

GUNSHOT WOUNDS OF THE INTESTINES, 1895–1901. (*Continued.*)

SEX. AGE.	LESIONS FOUND.	TREATMENT.	RESULT.	AUTHOR.	REFERENCE.
47. M. 52.	Wound of left lung and penetrating wound of abdomen.	No op.	Death in 8 hrs.	Richard Douglas.	——— 1900.
48. M. 46.	Ileum entirely severed, 10 perfors. of intestine, and several of mesentery.	Op. 4 hrs.	Death in 6 days from leakage of Murphy button.	"	——— 1901.

GUNSHOT WOUNDS OF THE LIVER, 1895–1900.

SEX. AGE.	LESIONS FOUND.	TREATMENT.	RESULT.	AUTHOR.	REFERENCE.
1. M. 24.	Lower margin of liver completely severed; perfor. of intestine.	Op. 24 hrs. No shock.	Recovery.	H. B. Palmer.	J. Med. and Sci., 1899–1900, VI.
2. M. 51.	Perfor. of liver, stomach, and kidney.	Op. 12 hrs. Shock.	Death in 24 hrs. p. op.	Glantenay and Neven.	Bull. et Mém. Soc. Anat., 1899, LXXIV.
3. M. Adult.	Perfor. of liver and ascending colon.	Op. 8 hrs. p. tr.	Death in 40 hrs. p. tr.	C. S. Miller.	Amer. J. Surg. and Gyn., 1899, XIII.
4. M. 12.	Perfor. of liver and small intestine.	Op. 6 hrs. No shock.	Recovery 1 month.	R. W. Winslow.	Annals Surg., 1898.
5. M. 16.	Perfor. of liver.	Op. at once.	Recovery.	———	Jahresb. d. Chir. Abth. d. Spital Basel, 1898, 58.
6. M. Adult.	Autopsy: laceration of liver.	No op.	Death in 5 days p. tr.	C. L. Heizmann.	Surg. Gen. Rep., 1895.
7. M. Adult.	Laceration of liver.	Op. 1½ hrs. p. tr.	Recovery 1 month.	Elliott.	"
8. M. 30.	Wound of liver.	No op.	Death in 3 weeks from abscess and sepsis.	R. Douglas.	———

GUNSHOT WOUNDS OF THE SPLEEN, KIDNEY AND BLADDER, 1895–1901.

	SEX. AGE.	LESIONS FOUND.	TREATMENT.	RESULT.	AUTHOR.	REFERENCE.
Spleen.	1. M. 28.	Perfor. of spleen.	Op. 12 hrs. Shock.	Death in 2 days p. op.	M. Peyrot.	Bull. et Mém. d. l. Soc. Chir., Paris, 1895, XXI.
Kidney.	1. M. Adult.	Wound of right kidney, and probably of intestine.	No op. No shock.	Recovery 2 months.	Schöffer.	Deutsch. Milit.-Arztl. Zeitschr., 1896, XXV.
	2. M. 20.	Perfor. of left kidney and spleen.	Op. 5 hrs. No shock.	Recovery 1 month.	Tiffany.	Tr. S. Surg. and Gyn. Soc., 1895, VII.
	3. M. 40.	Wound of right kidney and liver.	Op. 20 hrs.	Recovery.	R. Douglas.	——— 1901.
Bladder.	1. M. Adult.	Perfor. of bladder.	Op. 2 hrs. p. tr.	Death in 41 days p. op.	Haga.	C. R. XII Cong. Internat. d. Méd., 1899, V, pt. III.

SEX.	AGE.	LESIONS FOUND.	TREATMENT.	RESULT.
1. M.	50.	Bird-shot wound of colon, and kidney.	Op. 6 hrs.	Recovery.
2. M.	28.	5 perfors. of small intestine.	Op. 48 hrs.	Death in 56 hrs.
3. M.	Adult.	Wound of diaphragm and stomach.	Op. 12 hrs.	Death.
4. M.	30.	Wound of liver.	No op.	Death in 3 wks. from abscess and sepsis.
5. M.	19.	13 perfors. of small intestine.	Op. 36 hrs.	Death in 6 hrs. p. op.
6. M.	12.	4 perfors. of intestine and wound of internal iliac vein.	Op. 6 hrs.	Death on table from hemorrhage.
7. M.	45.	Wound of right lung, liver, portal vein, transverse colon, small intestine and sigmoid flexure.	Op. 4 hrs.	Death in 5 hrs. p. op.
8. M.	Adult.	Perfor. of transverse colon and liver.	Op. 18 hrs.	Death in 12 days from pneumonia.
9. M.	16.	Multiple perfor. of small intestine and liver.	Op. 30 hrs.	Death in 3 days.
10. M.	22.	Multiple perfor. of small intestine and liver.	Op. 25 hrs.	Death same night.
11. M.	7.	1 perfor. of jejunum; general peritonitis.	Op. 52 hrs.	Death in 3 days.
12. F.	—	3 perfors. of small intestine, 1 of colon.	Op. 4 hrs.	Recovery.
13. M.	21.	4 perfors. of small intestine, 1 of colon.	Op. 3 hrs.	Death in 13 days.
14. M.	17.	2 perfors. of omentum, 2 of colon.	Op. 2 hrs.	Recovery.
15. M.	20.	4 perfors. of intestine.	Op. 4 hrs.	Recovery.
16. M.	29.	6 perfors. of intestine, 1 of small mesenteric artery.	Op. 2 hrs.	Recovery.
17. F.	15.	Laceration of 5 ins. of ileum, severed and torn from mesentery. Protruding through large abdominal wound.	Op. 3 hrs.	Recovery.
18. F.	20.	Wound of descending colon.	Op. 3d day.	Recovery.
19. M.	40.	Wound of liver and kidney.	Op. 20 hrs.	Recovery.
20. M.	52.	Wound of left lung and penetrating wound of abdomen.	No op.	Death in 8 hrs.
21. M.	46.	Ileum entirely severed, 10 perfors. of intestine, several wounds of mesentery.	Op. 4 hrs.	Death in 6 days from leakage of Murphy button.

PENETRATING STAB-WOUNDS OF THE ABDOMEN, 1895–1901.

SEX.	AGE.	LESIONS FOUND.	TREATMENT.	RESULT.	AUTHOR.	REFERENCE.
1. M.	44.	Wound of cecum and mesentery.	Op.	Death in 3 days.	A. Baldwin.	W. Lon. Med. J., 1901, VI.
2. M.	28.	Protrusion of omentum; wound of liver, pleura, diaphragm; emphysema.	Op. 5 hrs. p. tr.	Recovery in 26 days.	H. Girard.	Bull. et Mém. d. l. Soc. Chir., Paris, 1901, XXVII.
3. M.	30.	Protrusion and strangulation of intestine.	Op. Shock.	Recovery in 3 wks.	"	Bull. et Mém. d. l. Soc. Chir., Paris, 1901, XXVII.
4. M.	40.	Perfor. at junction of mesentery with colon.	Op. 6 hrs. p. tr.	Recovery.	Auvray and Rochard.	Bull. et Mém. d. l. Soc. Chir., Paris, 1901, XXVII.
5. M.	34.	Multiple perfor. of intestine.	Op. 20 hrs. Shock.	Death 8 hrs. p. op.	Paul M. Rondani.	Ploies Pén. d. l'Abd., Bordeau, 1900.
6. M.	25.	Protrusion of omentum.	No op.	Recovery in 26 days.	"	Ploies Pén. d. l'Abd., Bordeau, 1900.
7. M.	27.	Perfor. with hernia of omentum and mesentery.	Op. 1½ hrs. No shock.	Recovery in 20 days.	"	Ploies Pén. d. l'Abd., Bordeau, 1900.
8. M.	24.	Fecal matter escaped from wound 2d day and continued 3 weeks.	No op. Severe shock.	Recovery.	"	Ploies Pén. d. l'Abd., Bordeau, 1900.
9. M.	35.	Perforation of intestine.	Op. 40 hrs. Shock.	Death 6 hrs. p. op.	"	Ploies Pén. d. l'Abd., Bordeau, 1900.
10. M.	35.	Perforation of intestine.	No op. Severe shock.	Death 24 hrs. p. tr.	"	Ploies Pén. d. l'Abd., Bordeau, 1900.
11. M.	42.	Perfor. of small intestine, prolapse and incarceration.	Op. during shock.	Death 12 hrs. p. tr.		Jahresb. d. Chir. Abt. d. Spital Basel, 1898.
12. M.	—	Intestine and liver cut.	Op.	Recovery.	M. F. Irving.	Br. Med. J., 1898.
13. —	—	2 wounds of tr. colon and small intestine.	Op. 1 hr. p. tr.	Recovery.	J. W. Chambers.	Md. Med. J., June 11, 1898.
14. —	—	Wound of small intestine.	No op. No shock.	Death in 12 hrs.	Duer.	Ind. Med. Rec., April 1, 1898.

PENETRATING STAB-WOUNDS OF THE ABDOMEN, 1895–1901. (*Continued.*)

SEX.	AGE.	LESIONS FOUND.	TREATMENT.	RESULT.	AUTHOR.	REFERENCE.
15. M.	45.	Protrusion of omentum.	Op. 1 hr. Slight shock.	Recovery.	J. P. J. Bush.	Lon. Lancet, March 20, 1898.
16. M.	15.	1½ ft. of intestine protruding.	Op. No shock.	Recovery in 19 days.	G. Bidie.	Br. M. J., Sept. 24, 1898.
17. M.	32.	Perfor. of descending colon.	Op. 5 hrs. Shock.	Recovery in 5 wks.	J. L. Wiggins.	Med. Rev., St. L., June 18, 1898.
18. M.	40.	No perfor. of intestine.	Op. 2 days p. tr.	Recovery in 40 days.	A. B. Johnson.	N. Y. Med. J., Sept. 17, 1898.
19. M.	22.	Perfor. of small intestine and mesentery.	Op. 23 hrs. p. tr.	Death in 4 days.	Fritz Breuer.	Inaug.-Diss., Bonn, 1897.
20. M.	25.	Prolapse and wound of omentum.	Op. at once.	Recovery in 3 wks.	G. Lotheiser.	Wien. kl. Woch., 1897, X.
21. F.	44.	Perfor. of tr. colon and omentum; escape of feces.	Op. at once.	Recovery in 28 days.	Gézav. Nagy.	Centr. f. d. ges. Hielk., 1897, XV.
22. M.	Adult.	Protrusion; wound of intestine 2½ ins.	Op. 12 hrs. p. tr.	Recovery in 17 days.	T. Wilcox.	Surg. Gen. Rep., 1895.
23. M.	34.	Protrusion; 2 wounds of intestine.	Op. at once.	Recovery in 23 days.	J. D. Glennan.	"
24. M.	17.	Perfor. of stomach.	Op. 2 hrs. No shock.	Recovery in 15 days.	Paul M. Rondani.	Ploies Pén. d. l'Abd., Bordeau, 1900.
25. M.	45.	Perfor. of stomach.	No. op.	Death 48 hrs. p. tr.	"	Ploies Pén. d. l'Abd., Bordeau, 1900.
26. M.	Adult.	Bayonet wound through entire stomach.	No. op.	Recovery in 20 days.	Surman.	Arch. gén. de Méd., 1897, VIII.
27. F.	8.	Protrusion of entire stomach and part of duodenum through wound.	Op. 2 hrs. Shock.	Recovery.	C. Buchanan.	Lon. Lancet, Aug. 23, 1897.
28. M.	37.	Incised wound of liver and perfor. of tr. colon.	Op. 1 hr. p. tr.	Death 3 days p. tr.	J. L. Wiggins.	
29. M.	18.	Incised wound of liver.	Op. Shock.	Recovery.	"	Jahresb. d. Chir. Abt. d. Spital Basel, 1898.

PENETRATING STAB-WOUNDS OF THE ABDOMEN.

DEFINITION.—Under this head are considered all penetrating wounds of the abdomen other than gunshot.

ETIOLOGY.—Wounds of this character may be inflicted with a knife, bayonet, poniard, such instruments being of various shapes and sizes; with pointed objects of various character, which produce a punctured or lacerated wound.

PATHOLOGY.—The appearance of the parietal wound depends altogether upon the character of the vulnerating body. Those wounds made with comparatively blunt instruments are usually lacerated, ugly, gaping wounds. Smoothly incised wounds bleed freely; their edges may rest in apposition or be widely retracted; wounds that sever the fibers of the parietal muscles transversely are gaping. In large wounds of the abdominal wall, whether incised or lacerated, their penetrating character is usually evident from the protrusion of a viscus, especially of the omentum, and, if perforating, from the escape of visceral contents. If the wound is inflicted with a small or sharp-pointed instrument, we can only judge of its possibly penetrating character by the length of the instrument and the force employed in inflicting the wound.

In ordinary stab or punctured wounds of the abdomen the viscera escape injury more frequently than in gunshot wounds. This is solely because of the length of the instrument used, and the effort at self-protection or withdrawal of the one wounded. In stab-wounds of the hollow viscera immediate extravasation depends upon the size and direction of the wound. Generally speaking, extravasation is greater in these wounds than in gunshot perforations. However, in small knife-wounds the visceral perforation is difficult of detection. In all perforating stab-wounds hemorrhage is usually a conspicuous feature; this is especially true of the solid viscera. The wound is, as a rule, limited in extent; thus we have single wounds of the liver, spleen, or intestines. Complicated wounds of this character are not frequent. When hemorrhage is profuse or extravasation

of visceral contents occurs, the subsequent pathological course does not differ materially from that following gunshot wounds. Hemorrhage, sapremia, and peritonitis are the complicating conditions. The omentum, liver, spleen, and kidneys are the organs most frequently wounded by stabs.

Symptoms.—It is of the utmost importance that we ascertain the character of the instrument used; its length, cutting edges, and breadth; blood-stains upon it are evidence of its depth of penetration; and from its size we may also infer the extent of visceral injury. Wounds inflicted with slender pointed instruments do not give rise to immediate symptoms, as a rule.

The shock manifest in stab-wounds of the abdomen is usually due to three causes: emotion, hemorrhage, and visceral injury. A large parietal wound with protruding viscera, even though there may be no perforation, gives rise to greater shock than a small perforating one of far more serious character. The hemorrhage, external and intraperitoneal, contributes largely to the degree of shock. Pain is sharp, cutting in character, but not otherwise significant.

Severe shock, griping pains, and muscular rigidity all suggest visceral perforation, yet the possible latency of these symptoms must not be ignored. In many small stab-wounds that are perforating in character the symptoms are all latent, and appear only with peritonitis. Langenbeck in his clinic recently presented a case in which there was a stab-wound of the abdomen with no symptoms for one week; then symptoms of perforation suddenly developed. At operation an incised wound of the stomach was found.

It is obvious that in incised wounds their penetrating character is usually more apparent than in gunshot wounds. It is also true that perforation occurs without symptoms. It therefore follows that in every case of stab-wounds of the abdominal parietes the wound must be investigated, if necessary enlarged, to determine its penetrating character; if penetrating, the abdomen should be opened to determine whether or not the viscera are injured. Opposed to such a course for explicit diagnosis I am aware that there are the highest authorities, yet I can not make any distinction in this respect between an incised and a gunshot wound. All symptoms subjective and objective

may mislead us. It is essential for diagnosis of perforation that we open the abdomen; it is imperative for proper treatment that we repair the injury. Granting that stab-wounds conservatively treated show a more favorable prognosis than gunshot wounds, it does not follow that this should cause us to deviate from the observance of the surgical precept that proper treatment is based upon correct diagnosis. The rankest conservative would not advocate inaction, if the intestines or stomach were known to be incised, or if hemorrhage was continuous from a wound of the spleen or liver; and as these effects of stab-wounds cannot be definitely known without exploration, the danger of uncertainty warrants the procedure.

PROGNOSIS.—Of 27 cases of stab-wounds of the abdomen surgically treated, 19 recovered and 8 died, a recovery rate of 70 per cent. In 6 cases expectantly treated, 3 recovered. These meager figures bear out the statement above made, that the fatality in stab-wounds is not as great as in gunshot wounds, when properly treated.

SURGICAL INDICATIONS.—Stab wounds of the abdomen should be handled just as are gun-shot wounds. The location of visceral injury after abdominal section is a much simpler matter in most cases, however.

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SUBPARIETAL INJURIES OF ABDOMINAL ORGANS.

DEFINITION.—We embrace under this head contusions, lacerations, and ruptures of the abdominal viscera without a penetrating wound.

GENERAL ETIOLOGY.—The occurrence of blows upon the abdomen, contusing or forcibly compressing its contents, is the most common cause of visceral injury. Circumstances incident to the violence materially influence the effect. Apparently the slightest violence may sometimes be followed by the most disastrous injury. Well-directed localized blows result in the greatest injury; as the kick of a horse or a severe punch. The direction of the blow counts for much; if perpendicular to the abdominal surface it is most harmful. Above the umbilicus the abdominal walls are thinner and offer less resistance than in the lower zone. The expectancy or preparedness of the individual may limit the effects of what otherwise might result in serious injury; the muscles are thrown into rigid defense, the entire body withdrawn and trunk flexed, costal margins approaching the ilia, and thus there is presented a firm cuirass of limited area to the vulnerating force. A muscular wall, therefore, offers greater resistance than a fat, pendulous one. The state of the hollow viscera, whether full or empty, to a large extent, determines their liability to injury; a distended stomach or bladder is less protected and more unyielding, and will rupture from less violence than an empty viscus. Pathological conditions affecting the parenchyma of solid viscera, ulcers and cicatrices that thin or weaken the wall of the stomach or intestines, predispose to rupture from the slightest causes. If a blow is received during sleep or when a person is intoxicated, or if inflicted suddenly upon one unawares, the elements of protection are absent, and the brunt of the injury is practically direct upon the viscera themselves.

Heavy weights passing over the body, as the wheels of a carriage or wagon; or crushing, as in buffer accidents; or forced

flexion, resulting in squeezing and compression, as in elevator accidents, are forms of great violence that rupture, displace, and pulpify the viscera. Slighter causes are responsible for not as extensive but equally as fatal accidents. Vomiting and straining at micturition under predisposing conditions may lead to rupture.

General Pathology.—The surface of the abdomen may show the marks of injury; abrasions, ecchymoses, or lacerations may show upon the abdominal wall. Subcutaneous rupture of the rectus abdominis may occur, as revealed by discoloration and irregularity of the surface.

Within the abdomen the effects of the injury present every conceivable variation. The peritoneum, its parietal, visceral, or mesenteric layers, may show multiple lacerations, or simply discoloration from contusion and extravasation of blood. Large blood-vessels, especially splenic, renal, and mesenteric, are found severed, and the cavity of the peritoneum filled with blood. Solid viscera are torn from their moorings, in part or entirely. Detached parts are found in remote places; or they may simply show laceration of greater or less extent. Where concussion is added to contusion the spleen and liver may be found completely disorganized, pulpified, though they may not have any visible rent. The intestines may be torn from the mesentery, ruptured, or severed entirely, their contents being extravasated into the cavity. Every grade of varying injury, from slight contusion to destructive maceration, may be found.

The immediate effects of the injury to the viscera are hemorrhage, escape of visceral contents and secretions, and, if the patient survives sufficiently long, the development of a localized or general peritonitis, assuming different pathological types according to the special conditions giving rise to it. These are demonstrable lesions, yet in reckoning the pathological results we must take into consideration the paralysis of the vasomotor center in the medulla, which is caused by the violent concussion of the sensory nerves. This nerve-commotion is symptomatically expressed in shock, and to this cause is attributed those cases of sudden death following blows upon the abdomen without visceral injury, unquestionably a very rare occurrence.

General Symptoms.—When it is known that contusions of the abdomen, unattended by visceral injury, sometimes produce

alarming symptoms, and when it is further appreciated that the surface appearance bears no relation to the extent of the internal injuries, it is admittedly difficult in any given case to answer the categorical question, Is there a visceral injury? The history of the accident is of the greatest importance in estimating its effects. The circumstances surrounding the patient at the time, his capacity or effort for resistance, the time of occurrence of the accident with relation to his last meal and last urination, the nature of the vulnerating body, the degree and direction of the force, the location of the injury, all these points have direct bearing.

The symptoms presented vary in intensity and modality, and will depend upon the time that has elapsed before the examination is made. The phenomena that attend rupture of the hollow viscera enable us to make an earlier and more positive diagnosis than in cases of injury to solid organs alone; but, as we shall see in the study of injuries of the special viscera, contusions generally produce multiple lesions, and the conspicuous symptoms of one may obscure all thought of another.

Pain: Presuming that the patient is seen immediately or soon after the occurrence of a severe contusion of the abdomen, we usually find him in pain, influenced in every case by the susceptibility of the individual and the degree of shock. Profound shock obtunds the sensibilities. The pain may be severe in the most superficial minor injury, or insignificant and transitory in the gravest visceral lesion. It may be local or general; it may be reflected; it may be only provoked by movement or pressure. It is sometimes described as tearing, as if something had broken loose, or burning; it is usually continuous and not paroxysmal. It is to be taken in evidence, but is never diagnostic.

Shock: After pain, the most appealing symptom is the general condition of the patient. Due allowance must be made for mental excitement, for fright and apprehension. Profound collapse may attend contusion in which there is no visceral injury. "I have seen marked collapse with no symptoms of rupture, patient shortly react, and be well in a few days" (Manley). Discount, as we must, intra-abdominal injuries by these accepted facts, yet the presence of shock, persistent and severe, must be regarded as strongly affirmative of visceral lesion. The absence

of shock for a time proves nothing. Literature abounds in cases of extensive intra-abdominal injury in which immediate shock was not present to any noticeable degree. Personal experience confirms this; yet exceptional cases do not constitute the rule, and in no class of accidents is shock so constantly present as in subperitoneal ruptures. The reasons for this are many; the mental attitude of the patient has already been spoken of; the violence is frequently received over the great sympathetic plexuses, hypogastric and solar; the often extensive and multiple injuries; the extravasation of foreign matter upon the sensitive peritoneum; lastly and chiefly, the hemorrhage which attends these lesions; one or all combined lower or suspend the vital functions. It is unfortunately true that the intensity of shock is not commensurate with the severity of the injury; it is also true that shock is not always immediate and pronounced. Sudden shock may be due to the nervous impression, alarming at the time, yet transient. Far more serious is it for the patient to show progressive shock; and if associated with acute anemia, recurring syncope, restlessness, and thirst, it indicates hemorrhage.

Shock denotive of subparietal visceral injury is expressed by the extreme pallor of the skin, the dull, lustreless eye, the dilated pupil, the pinched, drawn countenance, the shallow, irregular, sighing respiration, the cold extremities, the feeble, flickering or intermittent pulse, subnormal temperature, obtunded mentality, blunted sensibilities, and relaxed sphincters. Nausea and vomiting are rather constant symptoms.

In less profound shock the patient may be extremely restless, screaming with pain, even delirious, and calling for water. In some instances which I have observed, the pulse may be very slow and compressible, all other symptoms evincing unmistakable shock.

Physical Signs.—**Muscular rigidity** is a local sign of greatest value of intra-abdominal injury. The presence of general abdominal rigidity, associated with pain, tenderness, and shock, is especially significant of stomachal or intestinal rupture. In lacerations of solid viscera with attendant hemorrhage, rigidity is often absent. The accompanying injury to the parietes may cause the surgeon to be misled as to the presence of visceral injury unless he is on his guard.

If, soon after the receipt of an injury, free fluid is detected in the peritoneal cavity by varying or abnormal areas of dulness, and the patient shows evidence of acute anemia and shock, internal injury with hemorrhage cannot be doubted.

Sudden tympany with effacement of hepatic and splenic dulness, the physical sign of the rupture of a hollow viscus first pointed out by Morris, is fallacious and misleading. It is often absent; it is frequently too late in development for diagnostic purposes; distention of the colon may cause the same displacing resonance.

Meagre as the symptoms may appear, they are frequently all that are offered for diagnosis immediately after the accident. The further symptoms are not of the intra-abdominal injury itself, but of the effects or complications that arise therefrom. To await the development of these is to incur the hazard of infection and its increasing dangers. The diagnosis must be made within the first twenty-four hours. General symptoms are meagre and mythical (Michaux). All classifications are artificial and of no value (Le Dentu). Yet a knowledge of the accident, and the general resultant condition of the patient, aided as we are sometimes by specific symptoms indicative of injury of certain viscera, as hematemesis, melena, and hematuria, force upon us the necessity of definitely determining the nature and extent of the injury; and this can only be done through exploratory incision, the only certain means of diagnosing subparietal injuries of abdominal viscera, and the first step in their successful treatment.

PROGNOSIS.—Nimier, who strongly advocates the expectant treatment, has collected 307 contusions from the kicks of horses; 215 recovered, 92 died. Kirmisson says that when a man is kicked in the abdomen by a horse he has one chance in three of dying. Those dying, however, may be the only ones that receive internal injury. This is, perhaps, the most frequent causative accident, and it must be borne in mind that the blow is limited in area, often glancing, and rarely received in its full force; yet the mortality of about 30 per cent. shows the dangers of contusions from this comparatively moderate force. The rupture of hollow viscera is attended almost invariably by fatal result unless treated operatively. The general mortality of contusions of the

abdomen with rupture of viscera is placed at 96 per cent. In our table of collected cases we find 67 contusions of the abdomen subjected to operation, with 29 recoveries and 38 deaths, a recovery rate of 43.3 per cent. There were 46 cases treated expectantly; 37 died, 9 recovered, showing a recovery rate of only 19.5 per cent. under the expectant treatment, against 43.3 per cent. treated operatively.

Surgical Indications.—In the doubtful cases in which all knowledge of the accident is wanting, in which the symptoms are neither insignificantly mild nor alarmingly severe, it is advised to treat them expectantly (Berndt); to keep an armed expectancy; to keep them under observation for twenty-four hours (Bryant). In doubtful cases all text-books advise rest, opium, and limited diet, and qualify this by saying that if symptoms of internal rupture develop, then laparotomy is advisable. When symptoms of this appear it is generally too late for successful operation. Sir William MacCormac, in speaking of contusions of the abdomen, says that there is no condition in which the rule is more applicable, "When in doubt, operate." Operation, however, should not be undertaken heedlessly, but after judicial investigation of the history and symptoms. Severe pain and muscular rigidity, even without shock, call for operation. Hematuria, hematemesis, or melena, with pain and shock, urgently demand it. Ichel says that if the pulse increases out of proportion to body-temperature, operation should be done.

Against exploratory operation and its imaginary dangers Manley thus forcibly and erroneously expresses himself: "Surgeons should have the candor and honesty to confess that when perforation is absent opening of the abdomen, the disturbance of protective adhesions, the displacing of firm blood-clots, and opening of large, hidden, deeply-lodged blood-trunks may cut off the patient's only chance of recovery. . . . It is yet an open question whether we are ever justified in opening the abdomen for treatment of a hemorrhage within it produced by a contusion."

The intraperitoneal hemorrhage proclaims an intra-abdominal wound. The source of the bleeding and the special nature of the injury are entirely conjectural in most instances. We have to guide us only this general clinical fact: stomachal and intestinal ruptures do not bleed as freely as those of the mesentery

and solid viscera. From hemorrhage, primary or recurring, many of these patients die. It is surely not rational surgery to permit this disaster while we are quibbling over the propriety of opening the abdomen. Zeidler, in speaking particularly of lacerations of the liver, says that special symptoms do not arise until late, and that the danger is within the first twenty-four hours. This danger is hemorrhage. Carwardine's patient presented symptoms of shock from hemorrhage; he was pulseless and exsanguinated. The abdomen was quickly opened. "Rupture extended across the under surface from position of gall-bladder to posterior part, dividing the liver almost in twain." This patient was in perfect health in two months; without operation death was inevitable. McBurney's patient was in shock, the peritoneal cavity completely filled with blood-clots; ruptured spleen was found. Its removal was necessary to control the hemorrhage. Recovery was uneventful.

These are impressive illustrations of the necessity of quick interference in intraperitoneal hemorrhage from contusions. Years ago, in 1882, Marion Sims indicated the danger of extravasated blood into the peritoneal cavity. It is now known that if the patient escapes death by exsanguination the danger of sapremia awaits him, even though he may escape peritonitis. Shock due to hemorrhage does not contraindicate operation, but is a demand for it.

In all cases of contusion in which the phenomena that pertain to lesions of certain viscera appear, we are enlightened in our diagnosis and encouraged to undertake repair.

Then in conclusion, in all cases of subperitoneal visceral injury, diagnosed or strongly suspected, if the general condition of the patient permits, laparotomy should be done; and success will be in direct proportion to the promptness of the intervention.

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SUBPARIETAL RUPTURE OF THE STOMACH.

FREQUENCY.—The protection afforded by the costal margin, the thick walls of the stomach, its usually flaccid state, and its want of firm bony background are sufficient reasons to account for the infrequency of rupture of the stomach from contusions.

ETIOLOGY.—Well-directed blows, especially from the left side, engaging the stomach between the force and the vertebræ, are injuries most likely to be attended by rupture of the stomach. If the stomach is distended with food or gas at the time of the accident, it is more susceptible to injury. Morbid states of the walls of the stomach render it liable to rupture from slight violence. Overdistention from lavage in similar conditions may produce rupture.

PATHOLOGY.—Rupture of the stomach from contusion most frequently occurs along the greater curvature. The location of the rupture in those instances in which there is a predisposing element of previous mural disease is determined by the site of that disease. Rupture of the stomach rarely occurs without complication. The spleen and diaphragm are frequently implicated also. The entire wall of the stomach is usually rent; the peritoneal coat is less yielding than the inner linings. If the rupture is small the extravasation may be circumscribed, resulting in local suppuration. Spontaneous healing is not to be expected. Hemorrhage may be a prominent feature, though peritonitis is the usual cause of death.

SYMPTOMS.—Pain of a tearing, burning character usually attends the accident. Shock is generally immediate and pronounced. Vomiting is a constant symptom; blood generally appears in the vomitus; the absence of blood does not warrant the conclusion that the stomach is not lacerated. There may be hematemesis without complete rupture of the stomach-wall; in such instances we must take into consideration the nature of the accident and the associate symptoms. Muscular rigidity is usually marked. The diagnosis may be accurately determined by the inflation of

the stomach with air or with gas; if it responds by dilatation, it is reasonably certain that there is no perforation. If, on the other hand, the stomach cannot be distended, the gas escapes into the general peritoneal cavity, producing tympany with effacement of areas of normal dulness. The objection to the above diagnostic test is the straining and vomiting attending the passage of the tube, which, should rupture exist, would increase the extravasation. And, furthermore, the condition of the patient may preclude a measure so exhausting.

PROGNOSIS.—The violence is usually great, causing associate injuries which make prognosis extremely grave. Without operative intervention the mortality is practically 100 per cent. In our list we find 4 cases of rupture of the stomach; 2 subjected to operation recovered; 2 not operated upon died.

SURGICAL INDICATIONS.—This condition should be handled according to the principles already fully elucidated in the text.

SUBPARIETAL RUPTURE OF THE INTESTINES.

FREQUENCY.—Curtis found rupture of the small intestine in 112 out of 116 cases of visceral rupture from contusion. In 21 cases of traumatic rupture of the alimentary canal, Makins found the rent situated in the small intestines 16 times. Laplace says that the small intestine is ruptured in 75 per cent. of cases. In 89 cases of intra-abdominal injury, 23.59 per cent. were in some portion of the intestinal canal. In 113 cases of subparietal rupture of viscera that we have collected, 64 were of the intestines. In the order of frequency comes the ileum first, then jejunum, colon, duodenojejunal junction, and duodenum.

ETIOLOGY.—Rupture of the intestines usually follows well-directed violence, the percussion-force being applied to a small area. The determining cause of intestinal rupture is the engagement of the intestine between the vulnerating force and the posterior bony wall of the abdomen. In falls from a great height the duodenojejunal junction may be ruptured. Injuries to the sides of the abdomen or at the brim of the pelvis may cause rupture of the large intestine. Under general etiology we emphasized the various conditions predisposing to rupture.

PATHOLOGY.—The immediate cause of intestinal rupture is the compression of the gut against the bony parts, tearing or lacerating it, and so compressing it that necrosis with perforation follows soon after. In some instances the intestine shows a contused area, the actual rupture being some inches removed; in such cases the unequal tension of gaseous contents has caused the bursting of the intestines. The laceration may be linear, extending through several inches; circular, completely severing the gut; or a jagged rent; or the gut may be simply torn from its mesentery. Lacerations of the kidney are frequently associated with rupture of the colon, and in extensive buffer contusions we may expect to find complications in any viscus. Mesenteric rents, however, with rupture of large blood-vessels are the most frequent complication; and to this last injury is gener-

ally due the free hemorrhage, as uncomplicated intestinal rupture is rarely accompanied by much bleeding. The size of the opening usually permits some extravasation of intestinal contents, to which, though not necessarily extensive, the rapidly-developing peritonitis is due.

SYMPTOMS.—**Pain:** Pain is usually described as severe; it may be colicky or continuous and burning. Profound shock attends it. It is a constant symptom, though varying in intensity. It is generally referred to the umbilical region. In injuries of the colon it may be localized.

Shock: The nature of the accident, the seat of the blow, the condition and temperament of the patient, have their influence in the manifestation of shock. Profound collapse, from which there is no reaction, may be present. The degree of shock varies from this to severe, mild, or slight. Immediately following the accident appreciable shock may not be present, yet a few hours' observation will reveal it. The general condition of the patient is indicative of "abdominal shock." In severe cases we have subnormal temperature with irregular pulse. In all cases the pulse progressively increases in frequency and weakens. The respiration is significant; it is thoracic, shallow, and sighing.

Vomiting: Vomiting is a common symptom; it becomes of diagnostic value when it is persistent and intractable (Berndt).

Urinary Disturbance: In all intra-abdominal injuries, there may be partial or complete suppression of urine. Occasionally vesical tenesmus is complained of.

Obstipation: After a rupture of the intestinal tract there is usually an arrest of all bowel-action, and obstipation becomes a feature of the case. Bloody stools, therefore, if they appear at all, are too late for diagnostic purposes.

PHYSICAL SIGNS.—**Muscular Rigidity:** The abdomen is usually fixed, does not respond to respiratory movements, is frequently retracted. Muscular rigidity appears in fully 75 per cent. of the cases of intestinal rupture. Makins, Deaver, and Angerer insist upon its diagnostic value. Copious hemorrhage, profound shock, insensibility, and alcoholism antagonize its development.

Abdominal Tenderness: The blow may produce superficial injury sufficient to cause local tenderness. General hyperesthesia is common after any intra-abdominal accident. In intestinal

rupture we cannot rely upon local tenderness for guidance, either as to extent or seat of injury. Peritoneal inflammation, local or general, soon gives rise to tenderness. As an early symptom it is, therefore, of little value.

Splenic and Hepatic Dulness: I cannot attach any great importance to the much-talked-of sign of the disappearance of splenic and hepatic dulness. It is usually entirely absent. It appears so late that it is often of little value, and is frequently simulated by the meteorism of peritonitis.

Areas of Dulness: Usually the marked rigidity disguises the physical signs elicited by percussion. If hemorrhage is present sufficient in amount to produce relaxation, the free fluid may be detected in the cavity, or localized collections may be outlined by dulness. We must not confound this with the general dulness due to collapse of intestines and rigidity of the abdominal wall. Makins emphasizes the value of an area of impaired resonance, which he says is due to effusion and plastic lymph, producing early adhesions at the site of injury. This can only be of value several hours after the receipt of the hurt, and does not belong to the category of immediate symptoms.

Localized emphysema may develop in colonic or duodenal rupture.

SUMMARY OF SYMPTOMS.—The essential and most important information in the diagnosis of intestinal rupture is the history of the accident, embracing every point emphasized in the general symptomatology of subparietal injuries of abdominal organs. Pain, shock, and muscular rigidity are our most reliable symptoms, yet the examination must be painstakingly analytical, and the conclusions should rest upon the complexus and conformity of symptoms.

PROGNOSIS.—In 160 cases expectantly treated, 149 died, 11 recovered, 10 of which had fecal fistula (Angerer). In 36 cases of intestinal rupture due to the kicks of horses, 35 died (Chavasse). We may well ask, Was the thirty-sixth case correctly diagnosticated as rupture? Bryant reports 6 cases of rupture of the intestines, one of the duodenum; all died without abdominal section. Death in a few hours after the accident is due to shock or hemorrhage, later to peritonitis. Collier reports 2 cases of ruptured ileum in which death occurred three weeks after the

accident; these cases are notable for the long time the patients survived. Several instances are reported of patients dying suddenly in collapse from the fifth to tenth day. Of the 45 cases in our table subjected to operation, 18 recovered and 27 died; of the 19 treated expectantly, 5 recovered and 14 died. In the 5 recoveries without operation there must always exist a doubt of the correctness of diagnosis.

SURGICAL INDICATIONS.—Abdominal section should be resorted to immediately upon recognition of the injury, the condition of the patient permitting. The injured gut should be repaired by suture; resected, if necessary. All hemorrhage must be controlled, other viscera examined, abdominal cavity cleansed, preferably with dry gauze; irrigation should not be employed unless there is evidence of wide extravasation.

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SUBPARIETAL INJURIES OF THE LIVER.

FREQUENCY.—After the intestines, the liver suffers injury next in frequency from abdominal traumatism. Our table shows that of the 113 subparietal injuries of the abdominal viscera, in 20 the liver was involved.

ETIOLOGY.—Direct violence, kicks, blows, or heavy weights, as the wheels of a carriage passing over the region of the liver, result almost certainly in injury to its structure. In violent concussions, as in falls from a great height, the liver is broken from its attachments, its substance lacerated or disorganized. Squeezing, either by forced flexion of the trunk, as in elevator accidents, or by compression, produces the most extensive form of injury. The proximity of the liver to the parietes, its great size, fragile structure, and unyielding capsule are anatomical reasons rendering it especially susceptible to injury. In one instance muscular contraction alone resulted in injury of it (Waring). It must be appreciated that subparietal injuries of the liver may follow the slightest violence.

PATHOLOGY.—Injuries of the liver from contusions may involve the capsule or be entirely within the substance of the organ. Parenchymatous injuries, unaccompanied by apparent laceration, pursue invariably a fatal course; the arrest of the hepatic function and the shock of the accident usually cause immediate death. In other instances the subsequent pathological course is that of a diffuse hepatitis, probably suppurative, with an almost certainly fatal termination. Wounds of the liver bleed freely, and this is the most conspicuous feature in the pathology. The escape of bile into the peritoneal cavity in small amount is comparatively harmless, but when continuous it locally irritates and favors peritonitis, and its absorption produces cholemia. In rupture involving the gall-bladder or biliary ducts, the phenomena of icterus are marked. The associate injury to the peritoneum and to other viscera invites infection. We find as complications fractures of the ribs, diaphragmatic rents, laceration of the kid-

ney, ruptures of the small intestine and colon. Such associate injuries increase the immediate mortality, and gravely influence the ultimate prognosis. Of 135 cases of rupture of the liver, the right lobe alone was involved in 81.

SYMPTOMS.—The history of a direct blow or broad-side concussion, a knowledge of the nature of the accident, is of material aid in estimating the probability of hepatic injury. The recognition of a specific injury of the liver is very difficult, and the symptomatology is best considered in two stages, *immediate* and *remote*.

Owing to the immediate and continuous hemorrhage which accompanies these accidents, shock is generally pronounced. The immediate shock, if the violence is moderate, may be only slight, but its progressively deepening character is the salient feature of it. It is the shock of hemorrhage. The patient, therefore, becomes markedly anemic, is extremely restless—is thirsty—not infrequently delirious; as the shock increases he becomes relaxed, pupils dilate, his extremities and tongue become cold; he relapses into coma from cerebral anemia, and dies usually within a few hours after the accident. If the hemorrhage is less copious, reaction may occur. In this stage the patient is frequently transported to the hospital, or carelessly moved; then fresh hemorrhage occurs, as denoted by recurring syncope and relapsing shock.

Should the bleeding be temporarily arrested, our attention is now called to other symptoms. The pain may not have been a conspicuous feature; it depends largely upon the nature of the violence, and upon extraneous circumstances. In uncomplicated subparietal injuries of the liver the severity of the pain is an unimportant and variable symptom. There are two features connected with it that are somewhat diagnostic: it is intensified by vomiting, hiccough, or movement of the body, and influenced by respiration; it is referred to the right shoulder oftentimes. Vomiting, a symptom common to internal injuries, is usually present, but has no special significance. Hiccough is a serious symptom when it appears early; in many instances it suggests diaphragmatic injury; in three cases of wounds of the diaphragm and liver personally observed, distressing hiccough was an early symptom.

PHYSICAL SIGNS.—In addition to the general appearance denotive of shock, patients with injuries of the liver, perhaps more than with other intra-abdominal injuries, voluntarily restrain the respiratory movements. The abdominal muscles are fixed, and respiration is thoracic. Patients further aid in restraining movement of the liver by holding the side with the hand. Muscular rigidity, so characteristic of intestinal rupture, is not marked in these cases—indeed, it may be wanting. Local examination may be practically negative. If, however, the hemorrhage has been profuse, the presence of blood free in the peritoneal cavity may be detected, or it may accumulate, forming only a local area of dulness in the right lumbar and hypogastric regions.

As Zeidler very aptly says, rupture of the liver is very difficult to diagnose; the symptoms and physical signs in the first twenty-four hours are often insignificant, and this is the period of danger, as fatal hemorrhage may occur. After twenty-four hours symptoms may occur specific of hepatic injury.

The pain, local and referred, is more pronounced; there is definite local tenderness over the liver; and primary icterus from the absorption of bile from the peritoneum now appears. If the gall-bladder or its ducts have been ruptured, deep cholemia with bile-stained urine, persistent vomiting, and cerebral symptoms are marked. According to Edler, icterus appears in about one-fifth of the cases. Glycosuria is not a constant symptom of hepatic injury. After twenty-four hours the abdominal symptoms depend upon the amount of blood effused, and upon the rapidity of infection.

PROGNOSIS.—There are four undoubted cases of healed lacerations of the liver, hence the possibility of spontaneous recovery cannot be denied. Edler fixes the mortality of subparietal hepatic rupture at 85.7 per cent. Pirogoff, in speaking of wounds of the liver in general, asserted that in his experience they were always complicated and fatal. Expectantly treated, they are practically regarded as hopeless.

Contrast this with results obtained by operative interference. In 46 cases of traumatism of the liver and bile-ducts, 33 recovered, and 13 died, an operative recovery of 71.74 per cent. (Terrier and Auvray). In our table of 20 collected cases, 8 were

submitted to operation, with 5 recoveries and 3 deaths; of the 12 treated expectantly, only one recovered. Still, we must bear in mind that the occurrence of liver-cell emboli may set at naught the best efforts; the more severe and extensive the laceration, the more apt is such a misfortune to occur (de Forrest).

Dr. A. B. Johnson's patient appeared to be dying, was in extreme shock; abdomen was opened one and a half hours after the accident; belly was found full of blood; there was a ragged tear in the right lobe of the liver bleeding freely; the liver was partly torn from the diaphragm; there was also a transverse rupture in the right kidney. The patient expectorated blood, showing injury of the lung. Yet he recovered after operation.

In Heaton's case there was laceration of the liver, and a wound of the kidney necessitating nephrectomy. The patient recovered.

One of the most extensive wounds treated successfully by operation is recorded by Carwardine. In this case the liver was torn almost in twain, yet in two months the patient was well and strong.

Surgical Indications.—In view of the above results it cannot be questioned but that diagnosticated or supposed subparietal injuries of the liver should be treated by immediate abdominal section. Shock is no contraindication, unless the patient be actually dying. Schlatter reports 5 cases of wounds of the liver in which hemorrhage was controlled and wounds closed by suture; 2 recoveries. Cat-gut suture is no doubt the preferable way of dealing with these wounds, but such sutures are not always effective; even when tied over strips of gauze they may cut. If the wound can be securely sutured, and hemorrhage controlled, the abdomen may be closed without drainage, a very desirable point; yet we frequently have to resort to packing with gauze, which is to be preferred to the cautery or steam advocated by Doyen.

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SUBPARIETAL INJURIES OF THE PANCREAS.

FREQUENCY.—The remote location of the pancreas, protected by the ribs and overlying viscera, shields it from laceration and contusion. Uncomplicated injuries of it, therefore, are very rare.

ETIOLOGY.—Heavy weights passing over the abdomen, crushing in the ribs, are usually the cause of rupture of the pancreas. In 7 out of 10 cases this was found to be the character of the vulnerating force.

PATHOLOGY.—The pancreas may be simply contused, lacerated, or completely severed. The hemorrhage that attends the injury is profuse. Senn has shown experimentally that when the duct is severed, atrophy of the distal portion occurs, as it is not possible to restore the continuity of the pancreatic duct; this degenerative change may go on aseptically and without harmful results. Hemorrhage from the pancreas usually collects in the lesser peritoneal cavity, and if death is not immediate, we have a serohemorrhagic cystic accumulation, which is very different from a true traumatic pancreatic cyst, in which the peritoneum is not torn. The crushing character of the force usually produces a complicated injury; liver, spleen, kidney, intestines are frequently associated in the hurt. In 5 cases death was immediate in consequence of the extensive injuries.

SYMPTOMS.—Pain, shock, vomiting, the usual symptoms of abdominal peritonism, are present. There are no specific symptoms by which we can diagnose rupture of the pancreas without exploratory incision. Leith says that after the abdomen is opened it is necessary to use a Fergusson's speculum with electric light in order to obtain a view of the pancreas.

The symptoms by which pancreatic injury is recognized are the symptoms of the effect of the injury, occurring days or weeks afterwards. Emaciation is marked and rapid. Diarrhea is not mentioned. Perversion of appetite, observed in the cases of Hadra and of Brown, may be noticeable. In Hadra's case the boy could eat six or eight boiled eggs at one time; certain articles

of food were obnoxious to him; he detested table-salt. In several instances, a few days after the receipt of the injury a cystic tumor has been observed to develop. In Cushing's case the blow upon the abdomen was rapidly followed by the development of a pancreatic cyst, which closely resembled an aneurism. This cyst was drained five weeks after the injury; three days later there was a spontaneous evacuation of a subdiaphragmatic abscess. The patient recovered. Cushing found the specific gravity of the pancreatic fluid to be 1011, and not 1030 as is usually given. The daily quantity was about 500 c.c., and was very irritating to the epidermis. With rupture of the pancreas and with accumulation of its secretion and blood in the lesser omental cavity, the rapidly-developing tumor pushes the stomach before it, and the elastic swelling bulges between the colon and stomach. The relations of the cyst may be accurately demonstrated by inflation of the stomach and colon. Such accumulation can be readily mistaken for a true pancreatic cyst, as pointed out by Brown, Hadra, and others, and the escape through the foramen of Winslow could account for the formation of subdiaphragmatic abscess, or peritonitis. Of the 10 cases studied, in only one was there suppurative parotiditis.

Prognosis.—In contusions of the abdomen resulting in pancreatic injury, the violence is usually so great as to cause immediate death; otherwise, the cases pursue such a course as has been given above in the symptomatology.

Surgical Indications.—The impossibility of recognizing primarily injuries of the pancreas calls necessarily for treatment along general lines. If the indication of abdominal injury is sufficiently grave, the abdomen should be opened. Deep hemorrhage from the region of the pancreas should cause us to search carefully in that direction. The bleeding should be controlled by ligatures, and if the organ has not been completely severed, the breach closed by sutures. Packing with gauze may finally have to be resorted to. Provision must be made for drainage.

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SUBPARIETAL INJURIES OF THE SPLEEN.

FREQUENCY.—Of the solid viscera of the abdomen, next to rupture of the liver, laceration of the spleen from contusions occurs most frequently.

ETIOLOGY.—Its superficial position, especially when enlarged, its fragile texture, its tendency to engorgement, physiological and pathological, render it liable to rupture. A direct blow of moderate severity in the left hypochondriac or lumbar regions may suddenly force the spleen inward. The laceration may occur from the sudden displacement or from compression. In severe concussions of the abdomen, the spleen is found disorganized without an apparent wound. Spontaneous rupture of the spleen may occur after attacks of ague; Borrallier reports such a case.

PATHOLOGY.—Laceration of the spleen is frequently associated with rupture of the stomach; this complication has been found in 4 instances. It not infrequently is accompanied by laceration of the kidney; Weir and Abbe record such experiences. At autopsy portions of the spleen have been found detached, or the entire organ severed from its connections; the violence in these cases was usually great, death from shock and hemorrhage occurring immediately. Wounds of the spleen heal with wonderful rapidity. In a patient dying ten hours after an accident, Power found that the spleen had been torn on the anterior and external surface entirely through the capsule; the wound was closed by a firm white scar. In this case there was only slight hemorrhage.

SYMPTOMS.—The diagnosis of rupture of the spleen is made chiefly by the history of the accident, and the nature and seat of the blow, together with the symptoms of internal hemorrhage. The bleeding may be so profuse that the symptoms of shock from hemorrhage will mask all others. In less copious bleeding the blood collects in the left flank. Fixed dulness in the left loin is not present in intra-abdominal hemorrhage from other organs, the kidney not excepted. The progressive enlargement of this area of dulness, the shock, recurring syncope, restless-

ness, thirst, and vomiting, are symptoms characteristic of this lesion, and have been especially noticed by Ballance and by McBurney. The pain that attends this injury is not usually very severe; its reflection to the left shoulder is a significant feature.

Prognosis.—In extensive laceration death usually takes place within the first twenty-four hours from hemorrhage. In lesser tears the hemorrhage may be confined, and the complications of splenic abscess, septicemia, and peritonitis result. Rupture of a previously diseased spleen gives a better prognosis than rupture of a healthy one. Goffe records a most interesting case of contusion of the left side, followed by shock with retention of urine. The kidney was supposed to be injured and was treated expectantly. The patient did well and was allowed to sit up in three weeks. Six weeks after the injury, following a slight violence, the patient was seized with intense pain, cramps and vomiting, and became collapsed. Appendicitis was diagnosed. There was general plastic peritonitis; all the organs were examined, no pus accumulation was found. Later, septic symptoms appeared, and finally a subphrenic abscess of the right side was diagnosed. Dr. Wyeth operated by the transpleural route and evacuated a pint of pus, the patient dying four days after. The autopsy showed a necrosed spleen lying in an abscess-cavity. This case is mentioned in detail to illustrate the obscurity of symptoms, the liability to overlook injury even by so excellent a surgeon as Dr. Goffe, the course and complications that attend lacerations of the spleen, and finally the necessity of treating these cases by primary operation.

The general mortality of all wounds of the spleen is placed at 70 per cent. In subparietal lacerations of the spleen treated expectantly, death seems to be the invariable termination. We have collected 7 cases treated expectantly, all fatal. The operative mortality in injuries of the spleen complicated by wounds of the kidney or stomach is necessarily very high. Since 1890, 34 cases of splenectomy for rupture of the spleen have been reported, with a mortality of 41.2 per cent. (Warren).

Surgical Indications.—In injuries of the spleen the attendant shock is usually due to hemorrhage. The abdomen should be opened immediately. Simultaneously with operation transfusion should be employed. Wounds of the spleen may be sutured

and hemorrhage controlled, but in extensive lacerations *splenectomy* is infinitely the safer procedure.

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SUBPARIETAL INJURIES OF THE KIDNEY.

FREQUENCY.—Of 155 traumatic lesions of the kidney Keen found that 118 were subparietal. We have shown in the study of injuries of other viscera that renal injury is a frequent complication. In our table of cases we find it complicated with injuries of the liver, spleen, and colon; we have collected 11 cases of subparietal laceration of the kidney.

ETIOLOGY.—A direct blow in which the percussion-force is applied immediately over the kidney is the most common contusion resulting in injury to the kidney. Crushing force applied to the abdomen or to the lower part of the thorax usually produces laceration of the kidney and injury to the other viscera. Displacement with laceration occurs in falls from great heights.

PATHOLOGY.—The injury received may be merely a contusion without rupture of kidney-substance, or it may be a laceration involving the peritoneal surface, or the wound may be entirely extraperitoneal. The laceration may be limited to the cortical substance of the kidney, or it may involve the pelvis or ureter. The nature of the wound, whether or not it implicates the peritoneum, large renal vessels, or ureter, has direct bearing upon the symptoms and results of the injury. The direction of the wound to the axis of the kidney is usually transverse, that is, parallel to the direction of the tubules (Keen). The wound may be longitudinal through the whole extent of the kidney (Gunn). In subperitoneal wounds with laceration of the kidney-capsule, the hemorrhage escapes into the perirenal structures; and if the rent has involved a calix, urine will be mixed with the blood. When the peritoneum is torn, hemorrhage is usually free into the peritoneal cavity, the tendency being to extravasate in that direction rather than into the loin. If urine escapes along with the blood, peritonitis, plastic in character and not necessarily serious, will follow; but should the urine contain infectious matter, a fatal peritonitis will quickly supervene. In any event the kidney-wound with a peritoneal communication is always more

serious. It shows the degree of force and increases the probability of other visceral injuries. Anuria from shock may follow injury of the kidney, as we shall see later on; occasionally it is explained by there being only one kidney, an abnormality that must be taken into consideration in renal surgery. Morgagni says that when the kidney is severely lacerated the free hemorrhage from its substance so lessens blood-pressure that we have an arrest of renal secretion in the injured organ. In lacerations of the kidney the two important pathological phenomena are hemorrhage and infection, followed by suppuration and abscess-formation. We must bear in mind that hemorrhage through the natural channels from the kidney may occur without laceration of structure; that it is a phenomenon often due to disease and not to trauma. In one instance Morris found blood between the capsule and the cortex of the kidney, and yet there was no laceration.

Symptoms.—Contused wounds resulting in laceration of the kidney are usually the result of sufficient force to give rise to shock. Unless accompanied by hemorrhage, or otherwise complicated, the shock is not as prolonged or deep as in other visceral injuries. Nausea, with or without vomiting, is one of its conspicuous features. The shock is always greater when the injury is inflicted from in front, not because of the direction of the blow, but because of the fact that such forcible concussion results in other injuries, or in laceration of the peritoneal surface of the kidney, permitting free hemorrhage. Pain, as a rule, is severe, radiating downward to the genitals and thigh; there is usually retraction of the testicle. The immediate pain may be not very great, but should the pelvis become blocked with blood-clots, the accumulating blood and urine distending the kidney cause severe aching pain. The passage of blood-clots through the ureter gives rise to paroxysmal, colicky pains. Hemorrhage, as previously stated, is one of the important phenomena of renal wounds. There may be hematuria, or hemorrhage into the perirenal structures, or hemorrhage into the peritoneal cavity.

Hematuria is naturally regarded as an important symptom of renal injury, yet it must not be relied upon for diagnosis or treatment. If the ureter be severed, there will be no blood in the urine. If blood-clots block the pelvis or ureter, blood will be

absent; Newman refers to three cases in which there was a stricture of the ureter preventing hematuria. Hematuria may occur from simple contusions without laceration. It is a practical point in all wounds of the kidney that hemorrhage is suddenly arrested, and then begins again. In extensive lacerations, if Morgagni is correct in stating that there is suspension of the injured kidney's function, hematuria may be absent.

If the injury is extraperitoneal, and hemorrhage takes place about the kidney, a loin tumor with ecchymosis is quickly formed.

Intraperitoneal hemorrhage is recognized by the severity of the shock, by the marked anemia, by the presence of free fluid in the cavity, and by the development of an area of dulness which slowly changes with change of posture.

Difficult or frequent urination is often complained of. Anuria is attributable to the severity of the injury, to shock and hemorrhage; or to injury of both kidneys; or to the fact that there is only one kidney present and that one injured. We must eliminate the possibility of the bladder being full of blood in such cases. Local tenderness Newman thinks a diagnostic symptom of renal injury; in connection with other symptoms it is valuable. It may be due to injury of superficial structures only.

PROGNOSIS.—Immediate death is usually due to hemorrhage, or to injuries of other viscera. If patients survive the immediate shock of accident, recurring hemorrhage, septicemia, and peritonitis are dangers which bring the general mortality to about 50 per cent.

SURGICAL INDICATIONS.—The history of an injury and the appearance of hematuria are not sufficient to warrant interference. Such cases should be treated conservatively. If, however, the symptoms are threatening, there is marked shock and evidences of intraperitoneal hemorrhage or other injury, abdominal section should be done. The extent of the injury to the kidney will determine the propriety of *nephrectomy*, or suture and provision for lumbar drainage. Keen has shown the advisability of *primary* over *secondary nephrectomy*. We should choose the abdominal route only when we have reason to believe that there are intraperitoneal complications. In all other instances the kidney should be reached by lumbar incision.

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SUBPARIETAL INTRAPERITONEAL RUPTURE OF THE BLADDER.

FREQUENCY.—Since 1897, we are able to tabulate 9 cases of intraperitoneal rupture of the bladder out of 113 contusions of the abdomen, a relative frequency of 8 per cent. as compared with the total number of traumatisms.

ETIOLOGY.—"In 41 out of 107 cases of simple intraperitoneal rupture of the bladder it is distinctly specified or implied that the patient had been drinking alcoholic liquors, or was actually drunk at the time of the accident" (Rivington). The blunted consciousness, the excessive urinary secretion, and the relaxation consequent upon alcoholism, are the great predisposing causes of traumatic rupture of the bladder. Under such conditions only moderate external violence is necessary. Rupture from overdistention and muscular exertion is not likely to occur unless there is some previous disease of the bladder. A case falling under the observation of Morris had supposed rupture of the bladder; recovery under expectant treatment. Seven years later he had a severe attack of vomiting, went into collapse, and died of peritonitis. An autopsy showed a rupture at the site of the old cicatrix.

Crushing force, such as exerted by cart-wheels passing over the abdomen, may produce rupture of the bladder as well as other internal injuries. It may be due to fracture of the pelvic bones. Rupture occurs sometimes from exceedingly slight, overlooked violence, even when the bladder is not distended (Bryant).

PATHOLOGY.—The site of rupture is usually on the posterior surface near the fundus. The rent varies from a few centimeters to across the entire surface. Normal urine escaping from the bladder excites a non-septic plastic localized peritonitis. This fact has been experimentally proved by Wegner, Grawitz, and Waterhouse. Numerous autopsies confirm this. If septic matter is introduced along with the extravasating urine, or if there

is an associate injury of the intestine, then fatal septic peritonitis quickly follows. In Hamilton's and Ferraton's cases no sign of peritonitis could be detected. The wound shows but little tendency to heal. The collapsed and contracting bladder greatly lessens the size of the original rent. Coats thinks that the absorption of the urine by the peritoneum explains the absence of inflammation or healing in the wound, and that to the toxemia so produced death is usually attributable. Fracture of one of the pelvic bones is frequently an associate injury with intraperitoneal rupture of the bladder.

Symptoms.—The condition of the patient and the history of a contusion over the bladder will afford presumptive knowledge of injury of the bladder. The statement that something has given way, a tearing sensation, is often made. Following the injury the pain is of a burning character. More significantly diagnostic, however, is the urgent desire to urinate and the utter inability to do so. A few drops of bloody urine may be voided. The existence of shock will depend upon the degree of violence, the complicating injuries, and the condition of the patient at the time of injury. If intoxicated, he manifests but little shock. The amount of urine immediately extravasated also influences shock. The general symptoms may be so slight that the injury may be unsuspected for many hours. The diagnosis is further elucidated by the introduction of a catheter, and the failure to draw off urine. If a silver instrument is used it may pass through the rent in the bladder, and be felt against the parietes.

Tests by distension of the bladder with gas or fluid are misleading, and they are more dangerous than exploratory incision. For these reasons they should never be used. Exploration for intraperitoneal rupture of the urinary bladder is such a simple procedure that we should never hesitate to adopt it in doubtful cases.

Prognosis.—In 85 cases of simple intraperitoneal rupture, all died from the first to the sixteenth day—the majority in the first five days—showing the certain mortality of this injury. Heaton, in 1896, collected 37 cases of intraperitoneal rupture of the bladder treated by abdominal section and repair of the injury, with 15 recoveries. From 1897 to July, 1901, we find 7 cases of intraperitoneal rupture of the bladder subjected to operation, with 2 recoveries and 5 deaths; and 2 injuries not operated upon, both dying.

SURGICAL INDICATIONS.—If intraperitoneal rupture is diagnosticated, immediate abdominal section should be done, with repair of the rent and careful toilet of the peritoneum. Walsham, who has had 2 successful cases, and who has contributed excellent articles upon the subject, insists upon testing the wound by distention of the bladder with fluid before closing the abdomen.

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SUBPARIETAL RUPTURE OF THE THORACIC DUCT.

The rarity of this accident from contusions of the abdomen scarcely entitles it to consideration, yet the position of the duct against the vertebræ furnishes a background which will not yield to compressive force. Violence sufficient to reach the thoracic duct should apparently be at once fatal, and no doubt instances of its rupture have occurred, but we can find in literature only one recorded case of recovery following this injury, painstakingly and instructively reported by Dr. Thos. H. Manley (Phila. Med. News, Nov. 30, 1894).

The patient was a man 35 years of age, struck by the tongue of a wagon in the epigastrium, and run over by the wheels across the abdomen. There was but little shock, no vomiting, though tympany and sensitiveness developed quickly. In forty-eight hours he had every evidence of peritonitis. On the seventh day there was bulging over Poupart's ligament in the right side. Sixteen ounces of a milky fluid were evacuated by incision. The tumor was retroperitoneal, next to the sheath of the psoas muscle. The daily discharge was four to six ounces; it ceased by the tenth day. The specific gravity of the fluid varied from 1010 to 1019. It had a sweet taste, and looked like woman's milk. A careful microscopical and chemical examination was made of the fluid at various times on the same day and on successive days, and it was demonstrated to be pure chyle.

Dr. Manley thinks that there was not immediate rupture of the duct, but necrosis resulting in perforation and leakage. In concluding the report he says: "It certainly does seem extraordinary that a structure so well protected can sustain damage while overlying, more exposed parts escape."

The treatment of this case was wisely expectant.

SUBPARIETAL RUPTURE OF THE STOMACH, 1896–1898.

SEX.	AGE.	LESIONS FOUND.	TREATMENT.	RESULT.	AUTHOR.	REFERENCE.
1. F.	—	Perfor. of stomach near cardia.	Op. during shock.	Recovery.	Clubbe.	Austral. Med. Mag., 1898, XVII.
2. F.	19.	Perfor. of stomach.	Op. 30 hrs.	Recovery.	"	Austral. Med. Mag., 1898, XVII.
3. M.	22.	Autopsy: Rupture of stomach, small intestine and liver.	No op. No signs of internal injury.	Death 22 hrs. p. tr.	M. G. Rioblanc.	Cong. de Chir., Paris, 1897, XI.
4. M.	29.	Autopsy: Rupture of stomach.	No op. No sign of injury.	Death 40 hrs. p. tr.	W. G. McDonald.	Trans. Amer. Assoc. Obstet. and Gyn., 1896, IX.

SUBPARIETAL RUPTURE OF THE INTESTINES, 1896–1900.

SEX. AGE.	LESIONS FOUND.	TREATMENT.	RESULT.	AUTHOR.	REFERENCE.
1. M. Adult.	Autopsy: Multiple rupture of intestine and abdomen filled with feces.	No op. Collapse.	Death 5 days p. tr.	Frank Cross.	Guy's Hosp. Gaz., Lon., 1900, XIV.
2. M. Adult.	Autopsy: Rupture of sigmoid flexure, ½ inch.	"	Death 48 hrs. p. tr.	"	Guy's Hosp. Gaz., Lon., 1900, XIV.
3. M. 67.	Autopsy: Rupture of colon, 1½ inches.	"	Death 24 hrs. p. tr.	"	Guy's Hosp. Gaz., Lon., 1900, XIV.
4. M. Adult.	Abdominal wall rigid and tender.	"	Recovery in 5 days.	"	Guy's Hosp. Gaz., Lon., 1900, XIV.
5. M. Adult.	Abdominal wall rigid and tender.	"	Recovery in 4 days.	"	Guy's Hosp. Gaz., Lon., 1900, XIV.
6. M. 6.	Abdominal wall rigid and tender.	"	Recovery in 4 days.	"	Guy's Hosp. Gaz., Lon., 1900, XIV.
7. M. 33.	Autopsy: Rupture of posterior portion of duodenum at duodenojejunal junction.	No op. All the symptoms of peritonitis.	Death 4½ days p. tr.	A. Wm. Sheen.	The Clin. J., Lon., 1900, XV.
8. M. 35.	Tear of mesentery and vessels. Gangrene of colon, cecum, and lowest portion of ileum.	Op. 13 hrs. p. tr.	Death 10 mins. p. op.	"	The Clin. J., Lon., 1900, XV.
9. M. 16.	Autopsy: Rupture of duodenum, with escape of bile and feces.	No op.	Death in 5 hrs.	W. N. Belcher.	M. E. Hosp. Rep., N. Y., 1898.
10. M. 19.	4 perfors. of intestine. Tear in two places over 1 ft. in length.	Op. Shock.	Death in 24 hrs.	"	M. E. Hosp. Rep., N. Y., 1898.
11. M. 16.	Rupture of jejunum.	Op. in 24 hrs.	Death in 3 days.	"	M. E. Hosp. Rep., N. Y., 1898.
12. M. 32.	Rupture of jejunum.	No op.	Death in 2 hrs.	"	M. E. Hosp. Rep., N. Y., 1898.
13. M. 57.	Perfor. of small intestine. Abrasion of old hernial sac.	Op. Shock.	Death in a few hrs.	"	M. E. Hosp. Rep., N. Y., 1898.
14. M. 14.	Autopsy: Rupture of rectum, fracture of pelvis.	No op.	Death in a few hrs.	"	M. E. Hosp. Rep., N. Y., 1898.

SUBPARIETAL RUPTURE OF THE INTESTINES, 1896–1900. (*Continued.*)

SEX. AGE.	LESIONS FOUND.	TREATMENT.	RESULT.	AUTHOR.	REFERENCE.
15. M. 50.	Perfor. of jejunum.	Op. 5 hrs. Shock.	Recovery.	Clubbe.	Austral. Med. Gaz., 1898, XVII.
16. F. 21.	Perfor. of peritoneum.	Op. 5 hrs. Shock.	Recovery.	"	Austral. Med. Gaz., 1898, XVII.
17. M. 18.	Perfor. of ileum.	Op. 4½ hrs. Shock.	Recovery.	"	Austral. Med. Gaz., 1898, XVII.
18. M. 35.	Perfor. of duodenum.	No op.	Death in 70 hrs.	A. Schmitt.	Münch. med. Woch., 1898, XLV.
19. M. 27.	Rupture of duodenum and jejunum.	Op. 16 hrs. Severe shock.	Death in 24 hrs.	"	Münch. med. Woch., 1898, XLV.
20. M. 45.	3 perfors. of small intestine.	Op. 17 hrs. Severe shock.	Death in 6 hrs.	"	Münch. med. Woch., 1898, XLV.
21. M. 29.	2 perfors. of ileum and mesentery.	Op. 60 hrs. Severe shock.	Death in 12 hrs.	"	Münch. med. Woch., 1898, XLV.
22. M. 31.	Perfor. of small intestine and mesentery.	Op. 4 hrs. Severe shock.	Death in 6 days.	"	Münch. med. Woch., 1898, XLV.
23. M. 26.	Perfor. of small intestine and mesentery.	Op. 9 hrs. Severe shock.	Recovery in 60 days.	"	Münch. med. Woch., 1898, XLV.
24. M. 55.	Perfor. of duodenum. Incarceration of preëxisting hernia.	Op. 8 hrs. Severe shock.	Death in 4 days.	"	Münch. med. Woch., 1898, XLV.
25. M. 31.	Old incarcerated hernia, 55 cm. of ileum detached from mesentery; rupture of mesenteric artery and vein.	Op. 5 hrs. Severe shock.	Death in 4 hrs.	Madelung.	Deutsch. med. Woch., Berlin, 1898, XXIV.
26. M. 27.	Bowel wall torn through.		Death in 25 days p. tr.	W. H. Battle.	Lon. Lancet, Dec. 10, 1898.
27. M. 11.	Large tear in mesentery.	Op.	Recovery.	M. Sheild.	Br. Med. J., 1898, p. 1339.
28. M. 14.	Large tear of mesentery and jejunum.	Op. 3 hrs.	Death 8 hrs. p. op.	"	Br. Med. Jr., 1898, p. 1339.

SUBPARIETAL RUPTURE OF THE INTESTINES, 1896–1900. (*Continued.*)

Sex.	Age.	Lesions Found.	Treatment.	Result.	Author.	Reference.
29. F.	61.	Ecchymosis on posterior surface of bladder. Autopsy: Perfor. of cecum with extravasation of feces.	Op. during shock.	Death 4 days p. tr.	M. Shield.	Br. Med. Jr., 1898, p. 1339.
30. M.	22.	7½ inch tear of abdominal wall, 6 or 7 coils of intestine protruding.	Op. Shock.	Recovery in 5 days.	J. R. Jenkins.	Lon. Lancet, June 28, 1898
31. M.	Adult.	Laceration of intestines.	Op. sev. days p. tr.	Death.	Harvey Reed.	J. A. M. A., June 4, 1898.
32. M.	—	Admitted 10 days after injury.	No op.	Death on 11th day.	J. T. Rogers.	Med. Rec., Oct. 8, 1898.
33. M.	37.	2 perfors. of small intestine.	Op. 3 days p. tr.	Death immediate.	"	"
34. M.	25.	2 perfors. of jejunum.	Op. 22 hrs. Severe shock.	Death 13 hrs. p. op.	"	"
35. M.	35.	No external injury. Probably rupture of intestine.	No. op.	Death 3 days p. tr.	"	"
36. M.	25.	2 perfors. of small intestine.	Op. 33 hrs. p. tr.	Death 15 hrs. p. op.	C. O'Donovan.	N. Y. Med. J., Sept. 24, 1898.
37. F.	7.	Constriction and perf. of diverticulum.	Op. 15 hrs. Shock.	Death 1 hr. p. op.	H. N. Chapman.	N. Y. Med. J., Nov. 26, 1898.
38. M.	25.	Hernia formed by adhesion of omentum and transverse colon.	Op. 10 days p. tr.	Death during op.	G. Lotheisen.	Wien. klin. Woch., 1896, X.
39. M.	40.	Rupture of intestine.	Op. 11 hrs. p. tr.	Death 24 hrs. p. op.	P. Monclaire.	Bull. de la Soc., Anat., Paris, 1897, LXXII.
40. M.	—	Rupture of intestine.	Op. 20 hrs. p. tr.	Death 4 hrs. p. op.	"	Bull. de la Soc., Anat., Paris, 1897, LXXII.
41. M.	23.	Peritonitis.	No op.	Recovery in 30 days.	A. Yvert.	Archiv. de Méd. et de Pharm., 1897, XXIX.
42. M.	—	Peritonitis.	No op.	Recovery in 60 days.	F. Ende.	Archiv. de Méd. et de Pharm., 1897, XXIX.
43. M.	23.	Longitud. wound of colon.	Op. 21 days p. tr.	Recovery in 21 days.	Tédenot.	Cong. de Chir., Paris, 1897, XI.
44. M.	23.	Oblique rupture of small intestine, 1½ cm.	Op. 5 hrs. p. tr.	Recovery in 15 days.	"	Cong. de Chir., Paris, 1897, XI.

SUBPARIETAL RUPTURE OF THE INTESTINES, 1896–1900. (*Continued.*)

SEX. AGE.	LESIONS FOUND.	TREATMENT.	RESULT.	AUTHOR.	REFERENCE.
45. M. 30.	Rupture of small intestine.	Op. 4 hrs. p. tr.	Recovery in 19 days.	Tédenot.	Cong de Chir., Paris, 1897, XI.
46. F. 31.	Rupture of omentum.	Op. 10 hrs. p. tr.	Recovery.	"	Cong. de Chir., Paris, 1897, XI.
47. M. —	Longitud. rupture of cecum.	Op. 4 days p. tr.	Death 3 hrs. p. op.	"	Cong. de Chir., Paris, 1897, XI.
48. M. 41.	Perfor. of small intestine.	Op. 4 days p. tr.	Death 15 hrs. p. op.	"	Cong. de Chir., Paris, 1897, XI.
49. M. Adult.	Rupture of cecum.	Op. 6 hrs. p. tr.	Recovery in 26 days.	Chas. Fevrier.	Cong. de Chir., Paris, 1897, XI.
50. M. 55.	Rupture of great omentum and left renal vein.	Op. 3 hrs. p. tr.	Death 2 hrs. p. op.	Ariné Guniard.	Cong. de Chir., Paris, 1897, XI.
51. M. 28.	2 wounds of small intestine.	Op. 12 hrs. p. tr.	Recovery.	"	Cong. de Chir., Paris, 1897, XI.
52. M. Adult.	Perfor. of small intestine.	Op. 3 days p. tr.	Death 24 hrs. p. op.	P. Michaux.	Cong. de Chir., Paris, 1897, XI.
53. M. Adult.	Perfor. of colon, escape of 3 pts. blood.	Op.	Recovery.	"	Cong. de Chir., Paris, 1897, XI.
54. M. 30.	Laceration of mesentery, escape of 3 pts. blood.	Op.	Recovery.	"	Cong. de Chir., Paris, 1897, XI.
55. M. 33.	Laceration of mesentery, gangrene of loop of intestine.	Op. 23 hrs. p. tr.	Death 2 days p. op.	"	Cong. de Chir., Paris, 1897, XI.
56. M. 27.	Laceration of mesentery.	Op.	Recovery.	"	Cong. de Chir., Paris, 1897, XI.
57. M. 26.	Perfor. of intestine.	Op. 2 hrs. p. tr.	Recovery.	"	Cong. de Chir., Paris, 1897, XI.
58. M. 46.	Rupture of mesentery.	Op. 5 hrs. p. tr.	Recovery.	"	Cong. de Chir., Paris, 1897, XI.
59. M. 12.	Autopsy: Rupture of jejunum, entirely separated from duodenum.	No op. No signs of injury.	Death same day.	W. G. McDonald.	Trans. Amer. Assoc. Obstet. and Gyn., 1896, IX.

SUBPARIETAL RUPTURE OF THE INTESTINES, 1896–1900. (*Continued.*)

SEX. AGE.	LESIONS FOUND.	TREATMENT.	RESULT.	AUTHOR.	REFERENCE.
60. M. 36.	Autopsy: Transverse tear of ileum.	No op. No shock.	Death 18 hrs. p. tr.	W. G. McDonald.	Trans. Amer. Assoc. Obstet. and Gyn., 1896, IX.
61. M. 49.	Autopsy: Multiple rupture of intestines.	No op. No signs of injury.	Death 3 days p. tr.	"	Trans. Amer. Assoc. Obstet. and Gyn., 1896, IX.
62. M. 25.	Autopsy: Rupture of ileum.	No op. No signs of injury.	Death 4 days p. tr.	"	Trans. Amer. Assoc. Obstet. and Gyn., 1896, IX.
63. M. 37.	2 perfors. of ileum.	Op. 17 hrs. p. tr.	Death 8 days p. tr.	"	Trans. Amer. Assoc. Obstet. and Gyn., 1896, IX.
64. M. 19.	Tear in cecum, 1 inch.	Op. 1 hr.	Recovery.	"	Trans. Amer. Assoc. Obstet. and Gyn., 1896, IX.

Subparietal Injury of the Liver, 1897–1900.

Sex. Age.	Lesions Found.	Treatment.	Result.	Author.	Reference.
1. M. 15.	Autopsy: Rupture of liver and spleen.	No op.	Death 11 days p. tr.	A. W. Sheen.	The Clin. J., London, 1900, XV.
2. M. Adult.	Autopsy: Extensive laceration of liver.	No op. No shock.	Death 5 days. p. tr.	W. J. Gilbraith.	J. A. M. A., Jan., 1898.
3. M. Adult.	Constipation and ascites.	No op. No shock.	Recovery.	J. W. Mayo.	"
4. M. Boy.	Large quantity of bile in abdomen.	Op. 2 weeks p. tr.	Recovery.	"	"
5. M. 16.	Rupture of liver.	Op. 24 hrs. p. tr.	Death 8 hrs. p. op.	M. Sheild.	Br. M. J., 1898, p. 1339.
6. M. 3.	Rupture of liver, multiple fracture of ribs.	No op. Severe shock.	Death in a few hrs.	W. N. Belcher.	M. E. Hosp. Rep., N. Y., 1898.
7. M. 30.	Rupture of liver, spleen and right kidney.	No op. Severe shock.	Death in a few hrs.	"	M. E. Hosp. Rep., N. Y., 1898.
8. M. 43.	Rupture of liver, spleen, and colon.	No op.	Death 4 days p. tr.	"	M. E. Hosp. Rep., N. Y., 1898.
9. M. 35.	Rupture of liver, fracture of vertebræ and ribs.	No op.	Death soon p. tr.	"	M. E. Hosp. Rep., N. Y., 1898.
10. M. 35.	Rupture of liver and bladder.	No op.	Death 3 days p. tr.	"	M. E. Hosp. Rep., N. Y., 1898.
11. M. 3.	Rupture of liver and spleen.	No op.	Death soon p. tr.	"	M. E. Hosp. Rep., N. Y., 1898.
12. M. 27.	Rupture of liver.	No op.	Death soon p. tr.	"	M. E. Hosp. Rep., N. Y., 1898.
13. M. 62.	Rupture of liver, fracture of vertebræ and ribs.	No op.	Death soon p. tr.	"	M. E. Hosp. Rep., N. Y., 1898.
14. M. 42.	Rupture of liver and kidney.	No op.	Death 6 hrs. p. tr.	"	M. E. Hosp. Rep., N. Y., 1898.
15. M. 10.	Rupture of liver.	Op. Shock.	Death 12 hrs. p. tr.	"	M. E. Hosp. Rep., N. Y., 1898.
16. M. 60.	Rupture of liver.	Op. 6 hrs. p. tr.	Death on same day.	P. Monclaire.	Bull. de la Soc. Anat. 1897, LXII.

SUBPARIETAL INJURY OF THE LIVER, 1897–1900. (*Continued.*)

Sex.	Age.	Lesions Found.	Treatment.	Result.	Author.	Reference.
17. M.	—	Rupture of biliary ducts.	Op. 17 days p. tr.	Recovery.	P. Michaux.	Cong. de Chir., Paris, 1897, XI.
18. M.	39.	Rupture of liver.	Op.	Recovery.	"	Cong. de Chir., Paris, 1897, XI.
19. M.	16.	Rupture of hydatid cyst, not known to exist.	Op.	Recovery.	"	Cong. de Chir., Paris, 1897, XI.
20. M.	27.	Rupture of liver and omentum.	Op. 9 hrs. p. tr.	Recovery in 1½ months.	Ariné Guniard.	Cong. de Chir., Paris, 1897, XI.

SUBPARIETAL INJURY OF THE SPLEEN, 1897–1900.

SEX. AGE.	LESIONS FOUND.	TREATMENT.	RESULT.	AUTHOR.	REFERENCE.
1. M. Adult.	Autopsy: Rupture of spleen.	No op.	Death 24 hrs. p. tr.	Frank Cross.	Guy's Hosp. Gaz., Lon., 1900, XIV.
2. M. Adult.	Autopsy: Rupture of spleen and liver.	No op. No shock.	Death 24 hrs. p. tr.	"	Guy's Hosp. Gaz., Lon., 1900, XIV.
3. M. Adult.	Autopsy: Rupture of spleen, gall-bladder and kidney.	No. op. Severe shock.	Death soon p. tr.	"	Guy's Hosp. Gaz., Lon., 1900, XIV.
4. M. 25.	Rupture of spleen and blood-vessels.	Op. 5 hrs. Shock.	Death during op.	A. W. Sheen.	The Clin. J., London, 1900, XV.
5. M. Adult.	Rupture of spleen, liver and kidney.	No op.	Death 8 hrs. p. tr.	W. N. Belcher.	M. E. Hosp. Rep., N. Y., 1898.
6. M. 21.	Rupture of spleen and lung.	No op.	Death 24 hrs. p. tr.	"	M. E. Hosp. Rep., N. Y., 1898.
7. M. 35.	Rupture of spleen.	No op.	Death soon p. tr.	"	M. E. Hosp. Rep., N. Y., 1898.
8. M. 14.	Laceration of spleen.	Op.	Recovery.	Ballance.	Bos. M. and S. Jour., April 21, 1898.
9. M. 32.	Rupture of spleen.	No op.	Death 4 days p. tr.	P. Michaux.	Cong. de Chir., Paris, 1897, XI.
10. M. 26.	Rupture of splenic vein.	Op. 1½ hrs. p. tr.	Death 6 hrs. p. op.	Ariné Guniard.	Cong. de Chir., Paris, 1897, XI.
11. M. 19.	Rupture of spleen near hilus.	Op.	Recovery.	B. V. Beck.	Münch. med. Woch., 1897, no. 47.

SUBPARIETAL INJURY OF THE KIDNEYS, 1897–1900.

SEX. AGE.	LESIONS FOUND.	TREATMENT.	RESULT.	AUTHOR.	REFERENCE.
1. M. Adult.	Hematuria 5th day.	No op.	Recovery 12 days p. tr.	Frank Cross.	Guy's Hosp. Gaz., Lon., 1900, XIV.
2. M. Adult.	Hematuria and pyuria after 5 days.	No op.	Recovery 3 weeks p. tr.	"	Guy's Hosp. Gaz., Lon., 1900, XIV.
3. M. 3.	Hematuria for 24 hours.	No op.	Recovery 8 days p. tr.	"	Guy's Hosp. Gaz., Lon., 1900, XIV.
4. M. Adult.	No hematuria. Autopsy: Rupture of kidney.	No op. Shock.	Death on next day.	"	Guy's Hosp. Gaz., Lon., 1900, XIV.
5. M. 5½.	Hematuria 3d to 9th day. Rupture of kidney, pelvis filled by blood clot.	Op.	Recovery.	"	Guy's Hosp. Gaz., Lon., 1900, XIV.
6. M. 58.	Rupture of left kidney. Amputation of femur.	Op. Shock.	Death soon p. op.	W. N. Belcher.	M. E. Hosp. Rep., N. Y., 1898.
7. M. 18.	Internal hemorrhage and hematuria. Nephrectomy 18 days later.	Op. 33 hrs. p. tr.	Recovery in 3½ months.	Ariné Guniard.	Cong. de Chir., Paris, 1897, XI.

SUBPARIETAL INTRAPERITONEAL RUPTURE OF THE BLADDER, 1897–1900.

SEX. AGE.	LESIONS FOUND.	TREATMENT.	RESULT.	AUTHOR.	REFERENCE.
1. M. Adult.	Rupture of bladder.	Op. 4 days. Shock.	Death 24 hrs. p. op.	Frank Cross.	Guy's Hosp. Gaz., Lon., 1900, XIV.
2. M. Adult.	Rupture of bladder, general peritonitis.	Op. 3 days p. tr.	Death 24 hrs. p. op.	E. F. Robinson.	Surg. Gen. Rep., 1899.
3. M. Boy.	Rupture of bladder, fracture of pelvis.	Op. 24 hrs. p. tr.	Recovery 4 months p. op.	A. McLaran.	J. A. M. A., June 4, 1898.
4. M. Adult.	Rupture of bladder, urine in cavity; many hours before drainage. No peritonitis.	Drainage.	Death 1 wk. p. op.	J. W. Mayo.	J. A. M. A., June 4, 1898.
5. M. 53.	Rupture of bladder.	Op. Shock.	Death 10 hrs. p. op.	W. N. Belcher.	M. E. Hosp. Rep., N. Y., 1898.
6. M. 28.	Rupture of bladder.	No op. No shock.	Death 2 days p. tr.	"	M. E. Hosp. Rep., N. Y., 1898.
7. M. 26.	Rupture of bladder.	No op. No shock.	Death 3 days p. tr.	Lochboehler.	Personal Communication (1897).
8. M. 22.	Rupture of bladder, fracture of pelvis.	Op.	Recovery.	P. Michaux.	Cong. de Chir., Paris, 1897, XI.
9. M. —	Rupture of bladder, fracture of pelvis.	Op. 20 hrs. p. tr.	Death 12 hrs. p. op.	P. Monclaire.	Bull. de la Soc. Anat., 1897, LXXII.

1895–1901. 214 Abdominal Injuries.	Contusions.				Gun-shot.				Incised.				Total.	
	Operation.		No operation		Operation.		No operation		Operation.		No operation			
	R	*D*	*R*	*D*	*R*	*D*	*R*	*D*	*R*	*D*	*R*	*D*	*R*	*D*
Intestines	18	27	5	14	31	14	2	1	14	5	2	2	72	63
Stomach	2	0	0	2	7	3	0	0	2	0	1	1	12	6
Liver	5	3	1	11	4	2	0	1	1	1	0	0	11	18
Spleen	2	2	0	7	0	1	0	0	0	0	0	0	2	10
Kidneys	2	1	3	1	2	0	1	0	0	0	0	0	8	2
Bladder	2	5	0	2	0	1	0	0	0	0	0	0	2	8
Total	31	38	9	37	44	21	3	2	17	6	3	3	107	107

SUBDIAPHRAGMATIC ABSCESS.

DEFINITION.—Any collection of pus, or pus and gas, the whole or part of which intervenes between the diaphragm and the structures in contact with its lower surface (Godlee).

SYNONYMS.—Subphrenic abscess, hypophrenic abscess, pyopneumothorax subphrenicus, suppurative perihepatitis, perigastric abscess, suprahepatic abscess.

FREQUENCY.—Our familiarity with this disease as a surgical affection of the abdomen dates from 1879, when von Volkmann showed that an abscess situated below the diaphragm could be reached by a surgical operation. Leyden a year later gave us the classical symptoms for its recognition, and finally the monumental work of Maydl gave us a full comprehension of the etiology and symptomatology of the subject. Following these publications, numerous cases have been diagnosed and operated upon. The best contributions to the subject in American literature are from Weir, Berg, and Beck.

Concerning the frequency Carl Beck, in an article written in 1896, makes this prediction: "As soon as the profession at large will take the same interest in subphrenic abscess as they do now in appendicitis, the number of cases which in the whole present literature amounts to less than two hundred will rapidly swell to an enormous number." As compared to appendicitis it is infrequent, yet surgeons are alive to the possibility of it, and literature abounds in reports of cases.

ETIOLOGY.—We need only recognize the expanse of the diaphragm forming the dome of the abdomen, the viscera that are in relation, and the frequency of inflammation in them, to say nothing of infection from remote foci, to realize that subdiaphragmatic abscess may find its origin from a variety of causes. Maydl assigns the following: Mischief arising from—

The stomach,
The intestine,
The cecum and appendix,
Hydatids,

Subcutaneous injuries,
The liver and its ducts,
The kidney,
Metastases,
Wounds,
Various causes,
The ribs,
The thorax.

A few of these because of their frequency deserve special mention.

Gastric ulcer takes first rank as a cause of subphrenic abscess. A perforating ulcer, wherever located, may be followed by an encysted accumulation of pus, and it is to all practical intents a subdiaphragmatic abscess; ulcers located on the posterior wall especially produce this result. If the perforation occurs suddenly without preformed adhesions, then the visceral contents escaping free into the lesser peritoneal cavity set up a suppurative process—this is infrequent. Perforation of ulcers on the posterior wall is a gradual process, adhesions agglutinating the stomach to the pancreas, liver, and diaphragm, so that infection from the leaking stomach-contents is circumscribed. Under like conditions, ulcers of the duodenum and transverse colon may produce the typical subdiaphragmatic suppuration. When it occurs as a complication of appendicitis, the infection may take place in one of three ways: (1) by extension along the ascending colon; (2) retroperitoneally; or (3) in unusual instances, the appendix may occupy a high position under the liver.

Suppurative perihepatitis, secondary to hepatic abscess or of remote origin, gives rise to a typical subdiaphragmatic abscess. Perirenal abscess may likewise burrow in this direction, separating the peritoneum from the diaphragm. Diseased ribs and pyothorax may cause perforation of the diaphragm, when the subphrenic symptoms become more conspicuous than those of the preceding disease.

Severe contusions resulting in injuries to the kidneys, liver, pancreas, or spleen, causing hemorrhage, may be followed by infection and abscess-formation. Gunshot and stab-wounds followed by infection are not infrequent causes. One such case has

come under my observation, operated upon by the late Prof. W. T. Briggs.

Age: Exclusive of appendicitis, the causes that give rise to subdiaphragmatic abscess prevail in middle and advanced life; it is, therefore, of comparatively infrequent occurrence in childhood. Out of 176 cases collected by Lang, only 12 occurred in children. 3 of these were from appendicitis, 1 from simple gastro-enteritis, 2 from typhoid ulcers, and the remaining were of traumatic origin.

PATHOLOGY.—The epigastric, the right and the left hypochondriac are the regions concerned in subdiaphragmatic abscess. We may consider these abscesses under two grand divisions: (1) intraperitoneal, (2) extraperitoneal. The former is by far the more frequent. We may further consider them under the head of right and left subphrenic abscesses, the falciform and left lateral ligaments of the liver forming the dividing line in the intraperitoneal variety, and the attachment of the peritoneum to the diaphragm circumscribing and confining the extraperitoneal variety to the side from which it originates.

In intraperitoneal abscess of the left side the boundaries are fairly constant: in front, the anterior abdominal wall; behind and below, the left lobe of the liver adherent to the anterior abdominal wall, thus closing in the abscess-cavity at its anterior inferior angle; on the left, the cardiac end of the stomach, spleen, and left crus of the diaphragm; while on the right, the falciform and left lateral ligaments of the liver form the limiting internal wall, the arch of the diaphragm above completing the boundaries. These limitations apply to an abscess originating from perforation of the posterior wall of the stomach. The relations and boundaries of an intraperitoneal abscess depend entirely upon the etiological origin.

On the right side in intraperitoneal abscess the lower limiting wall is formed by the convexity of the liver; the falciform and lateral ligaments limit it internally; while the diaphragm forms its upper and outer boundaries. These limitations to an intraperitoneal subdiaphragmatic abscess, it must be understood, presuppose the formation of antecedent adhesions.

In the extraperitoneal variety we have the outer surface of the peritoneum forming the inner wall; and as this membrane is not

raised from the diaphragm by the abscess, these suppurations are limited in extent (Beck). In the large perirenal suppurations the parietal, but not the diaphragmatic, peritoneum yields to the accumulation. The perihepatic and appendicular inflammations account for the occurrence of extraperitoneal subdiaphragmatic abscess more frequently on the right side.

The most interesting pathological phenomenon connected with these subphrenic suppurations is the frequent appearance of gas as well as of pus in the abscess cavity—hence the name "pyopneumothorax subphrenicus." This gas may be due to the presence of a communication with some of the hollow viscera; but it is frequently found where no such communication exists, and is a product of decomposition due to the gas-producing bacillus. Pleural and pericardial effusions, pulmonary inflammation, and general peritonitis are associate complications or results.

Symptoms.—Subdiaphragmatic abscess we recognize not as a disease in itself, but as an accident or a complication of some preceding condition. Therefore, in the study of the symptomatology the symptoms of the causal disease must be taken into evidence, for they may be so pronounced as to cause us to overlook the development of the abscess. It must be understood that there is no one symptom or set of symptoms constantly present and pathognostic of subphrenic abscess. The very obscurity of its symptoms makes it at once interesting and dangerous. Its existence as extra- and intraperitoneal collections of pus, its development upon the one side or the other, or upon both, the presence or absence of gas, are conditions giving rise to varied symptomatic features. The physical signs must essentially depend upon the anatomical relations of the abscess. And, as we have seen, the frequent association of thoracic complications, pleural effusions especially, introduces an element for differentiation.

The development of a subdiaphragmatic abscess may be very sudden, with symptoms of intra-abdominal and thoracic distress pronounced from the first. Usually, however, it is insidious, and aside from the symptoms of general sepsis there are no special subjective symptoms. In the progress of the case confusion arises because of the existence of both thoracic and abdominal trouble. Leyden has pointed out the first and most important guide-post to a diagnosis, the existence of some previous intra-

abdominal trouble, and the absence of cough, expectoration, and pulmonary or pleural lesions to account for the physical signs. The substance of his idea is to determine by careful inquiry the history of the patient. In the majority of instances we can find the incriminating cause to be below the diaphragm. As we have seen in the etiology, it rarely proceeds from costal or pleural disease. Valuable as the history is, yet we know that the most frequent cause of subdiaphragmatic abscess is peptic ulcer, and we also know that this ulcer may exist and pursue a latent course, entirely unsuspected up to the time of perforation; and it is in this particular class of cases, in which there is an absence of history and an obscurity of symptoms, that the disease is overlooked. The frequency of this type renders it necessary for me to emphasize the existence; hence in any case of combined thoracic and abdominal symptoms, think of subdiaphragmatic abscess.

The general condition of the patient may be described as septic. In the more acute cases the temperature is irregular, often very high; recurring chills are frequent; colliquative sweats and delirium may be present; the patient bears a septic look, is extremely restless and uncomfortable. Microscopical examination of the blood will show marked leucocytosis; and even in the absence of septic symptoms, blood-examination should be made in this stage.

Pain is the only subjective symptom of significance. While this pain is usually felt in the epigastric or hypochondriac regions, it is often referred to the shoulder. We must bear in mind that this collection of pus is not in a normal cavity, but in one formed by inflammatory adhesions; therefore, the accumulating pus compresses surrounding parts. The pain is described as a heavy weight or oppression, embarrassing the function of adjacent viscera, limiting the action of the diaphragm, heart, and lungs.

Physical Signs.—Valuable as the facts above obtained may be, our diagnosis rests upon the physical signs, and in view of the protean picture of subphrenic abscess, the diagnosis can in no one case be considered easy. In a typical form of subphrenic abscess we have these physical conditions to contend with: the presence of gas within the abdomen high in the dome of the diaphragm;

beneath this the pus; above the gas is the thin expanse of the diaphragm, while within the thorax there is usually pleural effusion, compressed lung, and slightly displaced heart. So conspicuous are the thoracic phenomena that the clinical question is: Is it pyothorax, pyopneumothorax, or is it the "pyopneumothorax subphrenicus" of Leyden, that is, subdiaphragmatic abscess? Barlow was the first to solve these questions, and his conclusions were: (1) that the collection of gas was situated beneath a region in which the presence of pleural effusion was evident; (2) that pulmonary causes were absent which could have brought about a pyopneumothorax; (3) that it was difficult to suppose the possibility of such a separation between a fluid effusion above and gas below within the thorax; (4) that most distinct amphoric sound was present beneath the false ribs. To these Leyden has added the presence of normal vesicular murmur with normal fremitus at the apex, with a sharp line of differentiation from the amphoric breathing below; the very slight displacement of the apex beat; the rather inconsiderable bulging outward of the intercostal spaces.

Inspection: We observe fixed position of the trunk, with deep pain on change of posture, the patient preferring the shoulders well-elevated. The movements of the chest are not impaired. Respiration is thoracic, abdominal movements slight. The want of movement of the diaphragm Lauenstein emphasizes as a point greatly strengthening the suspicion of the existence of subphrenic abscess. Leyden emphasizes the absence of thoracic ectasia, and inversion of the intercostal spaces. Herrlich claims that bulging of the lower thoracic sphere is characteristic of subdiaphragmatic abscess. I should think that the presence or absence of this sign depends upon the amount and location of the pus-formation. In very emaciated individuals in whom we suspect subphrenic abscess on the right side, if we examine in a good light, we may detect the Bruce-Godlee sign, which is: if the pleura be healthy, we shall see the lower margin of the lung moving up and down during respiration. The truth of this observation may be tested by percussion and auscultation.

Palpation: In addition to the spontaneous pain mentioned, there is found an extreme sensitiveness to the touch along the arch of the ribs on the side on which the abscess is located. This

sensitiveness marks the adhesions limiting the abscess. Too, we notice tension of the recti in the epigastric region. We may further feel the spleen and liver displaced downward. There is retarded movement of the lower thorax. Tactile fremitus is diminished or lost over the lower part of the chest on the affected side. The apex beat is normal or slightly displaced ordinarily; if there is pleural effusion on the left side, there may be marked displacement. In large subdiaphragmatic accumulations on the right side without pleural effusion it may be displaced to the left. Leyden emphasizes the value of the normal or very slightly displaced apex beat, in differentiation from pleural effusion. If pus is in close proximity to the parietes, rigidity may be noticed; and localized edema, a sign always significant of underlying pus, may be detected.

Percussion: Percussion of the chest from above downward gives a note increasing in pitch and intensity above, and merging below into a full tympanitic sound; this only occurs in cases in which gas as well as pus is present. This expanse of tympany may extend up to the third rib. If, as is frequently the case, we have serous effusion in the pleural cavity, then we have dulness above, tympany below, and at a still lower level dulness. This is Barlow's diagnostic observation, differentiating a pyopneumothorax from subphrenic abscess; he says that in thoracic effusions it is difficult to conceive of gas at a lower level than the effusion. In those subphrenic abscesses in which gas is not present, the dulness reaches high up into the thorax, highest in front, fading off at the sides, and lowest behind, its shape being just the reverse of what a pleural effusion would be. The area of normal hepatic and splenic dulness is displaced downward.

Auscultation: Auscultation may show absence of respiratory murmur from the third rib downward. There is normal vesicular murmur with normal fremitus at the apex; below this there is amphoric breathing and metallic tinkling, and the bell-sound may be elicited. The sharp line of differentiation of the vesicular murmur from the amphoric breathing is one of the best diagnostic points. A loud friction-sound may be heard in the axilla, and even below the limits of the pleura. Breath-sounds may be heard beneath the false ribs. The heart-sounds are arhythmical, and displaced under certain conditions.

Exploratory Puncture: Where an abscess is suspected exploratory aspiration under strict aseptic precautions is advisable. However, we must bear in mind that pus may be present and yet the aspiration be negative; we may fail to strike the pus cavity with the needle, or the pus may be so viscid and thick that it will not flow. Fürbringer suggests that the motions of the needle are pathognomonic; this test has proved unreliable. If pus is withdrawn, and shows by microscopical and bacteriological examination a preponderance of the colon-bacillus, it is in all probability of intestinal origin.

DIFFERENTIAL DIAGNOSIS.—Clear as the symptoms may be, distinct and pronounced as the physical signs may appear, yet the diagnosis of a subdiaphragmatic abscess is none-the-less difficult. Many of the signs emphasized may be absent or difficult to obtain, and Leyden himself says that in the absence of history and definite etiology the diagnosis is often impossible. Pleural effusions, pyothorax especially, offer the greatest differential difficulties.

Subphrenic Abscess.	*Pleural Effusion.*
History.—Some intra-abdominal trouble.	Pleural or pulmonary disease.
Onset.—Usually gradual.	Sudden.
Respiration.—Not disturbed at first.	Markedly disturbed from the onset.
Movements of Chest.—Not impaired.	Diminished on the affected side.
Enlargement of Chest.—No pronounced ectasia.	Pronounced ectasia, with widening and bulging of interspaces.
Heart.—Normal or only slightly displaced.	Markedly displaced.
Percussion.—Tympany below normal limits of resonance; below this, well-defined dulness.	Absolute flatness all over the effusion.
Auscultation.—Breath-sounds may be heard below the level of dulness, descending with inspiration. Amphoric respiration, tympany, and the bell-sound.	Respiratory murmur is absent or greatly diminished. Amphoric respiration, tympany, and the bell-sound are all absent.

From *pyopneumothorax* subdiaphragmatic abscess may be distinguished by the history of pulmonary disease and respiratory symptoms in the former. The only difficulty arising would be an abscess associated with pleural effusion, and here we would find amphoric breathing underneath the floating ribs; and percussion would reveal dulness high in the chest, below this a tympanitic area, and still lower, dulness.

A non-gas-containing subdiaphragmatic abscess on the right side presents the greatest difficulties in distinguishing from hepatic abscess of the upper surface. The causes are so frequently the same that unless the liver-cells were found in the pus, the case might be operated upon without any definite idea as to the pathological condition.

PROGNOSIS.—The abscess, by perforating into adjacent viscera or discharging externally, may heal spontaneously; but by far the majority of cases treated expectantly die.

Of 74 cases treated surgically 39 lived. The causes of death were sepsis from insufficient evacuation, coexisting suppuration in adjacent organs, perforations of the pleura and pericardium, and, strange to say, very rarely peritonitis. The operative mortality is placed at about 50 per cent.; this is entirely too high if the diagnosis is made early. H. Graves reports six cases in which early operation was done, with five recoveries.

SURGICAL INDICATIONS.—The treatment is essentially surgical. There are no contraindications to operation unless the patient is actually dying. The essential indication is to provide free drainage. This may be obtained in one of two ways: (1) By the transpleural route. The pus is reached by excision of one or two ribs (8th, 9th, or 10th) in the median axillary line, opening the diaphragm from above; the danger in this method is of pulmonary collapse or infection of the pleura. Subphrenic abscesses should not be emptied by means of the transpleural operation in other cases than those in which an empyema exists, or in which the pleural cavity is partially obliterated. (2) Incision made beneath and following the line of the costal arch; the peritoneum is exposed but not opened; following this upward between the peritoneum and diaphragm until the adhesions which surround the abscess are reached, an opening is made into the abscess-cavity with blunt forceps.

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ANEURISM OF THE ABDOMINAL AORTA.

DEFINITION.—A tumor filled with blood, fluid or coagulated, communicating with the artery and confined by one or more coats of the vessel.

FREQUENCY.—Aneurism of the abdominal aorta is infrequent as compared with that of the thoracic. Of 468 cases of aortic aneurism at St. Bartholomew's Hospital, only 23 involved the abdominal aorta (Osler). Of 551 cases of aneurism collected by Crisp, 59 were of the abdominal aorta. Biggs found 4 abdominal aneurisms in 30 cases involving the aorta. Of aortic aneurism, that of the abdominal aorta occurs third in point of frequency, aneurism of the arch being most frequent, and of the descending portion next (Richter). Up to 1865, Lebert had collected 104 cases; from that date to 1881, Eger has collected 90 cases; since 1883, I have collected 73 cases.

ETIOLOGY.—**Age**: It is most common between 35 and 55 years. Aitken records an instance of an aneurism of the abdominal aorta of the size of a golf-ball in a boy nine years old. Gross refers to Mr. Armitage's case in a child seven and one-half years of age.

Sex: Sex has a most distinct bearing. It is variously estimated as from four to seven times more frequent in males. Of 59 abdominal aneurisms, Crisp found only 8 in women; his investigations ended in 1874. Since that time, Foot has been able to add only 9 cases in women.

Among the alleged causes of aneurism the late Surgeon-General Hamilton regarded climate as the only constant element. In a very exhaustively statistical article he showed that neither syphilis, alcoholism, heat, nor occupation influenced its development. He disproved the general statement that it is more common in northern climates, and found that 7.73 deaths per thousand in San Francisco were due to aneurism. Humidity and even temperature were the supposed requisite climatic conditions. Guiteras found it very common in Charleston, Eldridge remarked upon its frequency among the foreign residents in Japan; he at-

tributed it, however, to other causes. Rodman's investigations show it to be three times more common in the negro than in the white race.

The constant coexistence of atheroma and aneurism indicates a causal connection. Of 29 specimens of aneurism examined by Coats and Auld, in all atheroma was present. These authors maintain that the atheromatous change in the intima is the primary process, that the media yields from atrophy or rupture, and that aneurism occurs most frequently at that period of life in which the endarteritis, a senile change, is beginning, before the artery is strengthened by the secondary peri-arteritis, and while yet the individual is capable of great muscular exertion, which increases the blood-pressure. The older school, of which Recklinghausen was the head, regarded the atheroma merely a coincidence, and attributed the formation of aneurism to weakness in the media.

Heller maintains that aortic aneurism is the result of a syphilitic process. After drawing attention to such facts as its frequency in syphilitics, the occasional development of aneurism in both man and wife, and the good effects of anti-luetic treatment, he shows that the primary changes consist in a circumscribed or diffuse infiltration of round and giant-cells, involving the media primarily, along the vasa vasorum of which the process is most active; the muscular element undergoes more or less destruction, and secondarily the intima becomes involved, a process analogous to syphilitic lesions in viscera. These inflammatory changes are doubtless generally due to syphilis, but other infectious processes might account for them. It is a conspicuous fact, however, that aneurism is more common among syphilitics.

Muscular exertion can only produce aneurism in an artery predisposed by disease.

Pathology.—Fusiform, sacculated, dissecting, and false aneurisms are all described in connection with the abdominal aorta, but as the sacculated is the only form that admits of diagnosis or surgical treatment, we are concerned with it alone; and as only about 50 per cent. arise from the anterior aspect, it will be seen that a comparatively limited number are amenable to surgical treatment.

Any portion of the ventral aorta may be the seat of aneurismal development, but its most usual site is between the diaphragm and the point at which the superior mesenteric artery is given off. Not infrequently the celiac axis or its branches spring directly from the aneurismal sac.

The tumor, usually smooth and globular, may be irregular in outline and modified in its shape by the pressure of the vertebræ or ribs, or from the unequal deposition of clot. Early in the formation of an aneurism there is compensatory hypertrophy due to peri-arteritis. The tunica intima and tunica media have generally disappeared, and the tunica adventitia, together with newformed fibrous tissue, constitutes the thick wall of the aneurism. The resistance and hardness of the tumor are due to the thickness of its wall, and to the age and extent of coagulation. Aneurisms of backward evolution erode the vertebræ, disintegrate the ribs, and present posteriorly; whilst those of anterior growth, by their size and position, compress and displace the viscera.

Aneurism of the abdominal aorta may be associated with aneurisms elsewhere; Biggs found three aneurisms in one patient, two of the thoracic and one of the abdominal aorta; Prudden records an aneurism of the abdominal aorta associated with one of the right common iliac; Hinsdale exhibited a specimen of aneurism of the abdominal aorta, left common iliac and external iliac.

Spontaneous cure may occur by the coagulation and organization of the contents of the aneurismal sac and occlusion of the artery. Such a favorable course is not to be anticipated. The continuous growth of the aneurism may result fatally by its pressure-effects, by emboli producing peripheral gangrene; Horrock's patient had complete paraplegia of both legs, autopsy showing a small aneurism near the celiac axis and embolic occlusion of both iliacs, no gangrene resulting. The usual termination is in rupture, which may be due to the blood-pressure upon a weak portion of the sac unprotected by laminated clot, to necrosis of the sac-wall from pressure against vertebræ or ribs, or to infection and inflammation of the sac-wall from intestinal adhesions. Rupture is frequently precipitated by muscular exertion or external traumatism. Rupture may occur into the peritoneal or pleural cavities, or into the alimentary canal. The loss of blood under these circumstances is unrestrained, and death is

usually immediate. Subperitoneal ruptures are generally due to pressure; the aperture is small, a clot may partially occlude the opening, and the escaping blood is confined in the retroperitoneal tissue. The hemorrhage is, therefore, gradual in such cases, and the ultimate and inevitable death may be delayed many weeks. Cases have been reported of rupture into the thoracic duct, pelvis of the kidney, bile-channels, mediastinum, and pericardium. Death may occur from causes entirely remote from the aneurism; as such patients are the subjects of arteriosclerosis, cardiac lesions, cerebral hemorrhage, and nephritis are frequent complicating conditions, responsible for death.

Symptoms.—An aneurism of surprising dimensions may develop from the abdominal aorta without giving rise to physical or subjective expression of its existence. This is particularly true in aneurisms of backward growth, and in those involving the lower portion of the aorta. Foot has specially remarked upon the latency of symptoms of abdominal aneurism in the female; her abdomen, for anatomical and physiological reasons, is capable of keeping secret conditions which the male's would soon disclose. The cases, however, are exceptional indeed, which escape detection after scrutinizing examinations through any length of time. The development of an anterior sacculated aneurism gives rise to symptoms which are usually diagnostic.

Pain: The earliest, most impressive and persistent of all symptoms connected with aneurism of the abdominal aorta is pain, variable in intensity, character, and occurrence. It is sometimes local, more frequently referred. It is described often as a dull ache, as throbbing, as lancinating, and as cord-like. It is usually felt at the site of the pressure of the aneurism over the roots of the spinal nerves, and radiates through their distribution. It is said that in erosion of the vertebræ the pain is chiefly nocturnal and boring in character. This is not borne out by clinical observation; there are many cases in which the bodies of some vertebræ are almost absorbed, and the pain is insignificant. Though usually characteristically continuous, the pain may be intermittent or paroxysmal in character, and may occur chiefly at night, and be referred to the genitals and thighs, closely simulating renal colic; Chavasse's case had these features. Aneurisms in relation with the diaphragm give rise to pain in the thorax, shoul-

der, and neck (Biggs). Pain may occasionally be relieved by change of posture; Someren's patient was perfectly comfortable in a recumbent position. A surcease of pain for long intervals has been noted, possibly due to a change in the form and relations of the aneurismal tumor.

Abdominal Throbbing: This is a symptom complained of by many patients. It is described as a jumping, throbbing, quivering, or fluttering sensation in the abdomen; it is not persistent always—it may be felt only when standing, or after exertion, and one woman complained of it only before menstruation. This subjective symptom will only be present in rather large aneurisms, and usually when the objective pulsation is quite apparent.

Dyspnea: Dyspnea is a frequent symptom. It may be due to pressure, or to the size of the tumor restricting the movements of the diaphragm; it is also attributable, in many cases, to complicating cardiac distress.

Cough: Cough may be excited by pressure from below. Hemoptysis may occur from rupture into a bronchus.

Digestive Disturbances: From pressure, displacement, and distortion by adhesions, digestive symptoms may be conspicuous in the history of aneurism. Frequent vomiting is a fairly constant symptom. In Ewart and Jaffrey's case the symptoms were indicative of pyloric obstruction; when the abdomen was opened the stomach was found stretched over the top of the aneurism. A similar observation was made at Johns Hopkins Hospital. For eighteen months Mr. Maylard's patient had troublesome bowel-complaint, constipation, with occasional attacks of mucous diarrhea; and finally a pulsating swelling to the left of the dorsal spine disclosed the true nature of the case. The constipation which, in some instances, may be exceedingly obstinate, due either to constricting adhesions or to compression of the bowel by the sac, the associate vomiting and depression, and the presence of a tumor, may simulate fecal impaction. Blood may appear in the vomitus or in the stools; this is usually from rupture of the aneurism into the alimentary canal, and occurs only in the final scene.

Emaciation: Patients presenting the well-marked symptoms of agonizing pain, gastric and intestinal disturbances, very naturally lose flesh, have progressive muscular weakness, and evince

great anxiety of mind. But, in some instances, the emaciation is conspicuous beyond all other symptoms; this is strikingly illustrated in the recently reported case of Kiliani, in which his patient suffered with epigastric pain only after eating, yet he lost twenty-five pounds of flesh in a few weeks.

Motor and Sensory Disturbances: Muscular weakness, amounting to partial or complete paralysis, and sensory disturbances other than pain, remote from the seat of the aneurism, are occasionally referred to in the clinical records of cases. These symptoms occur in those aneurisms which, by pressure, involve the roots of the spinal nerves. Beevor's patient complained of burning and a numb feeling in the left thigh, as far down as the knee; there was progressive general weakness, but especially of the left leg, deficient tactile sensibility of the anterior surface of the left thigh and groin, and a striking loss of left patellar reflex as compared with the right.

PHYSICAL SIGNS.—**Inspection:** In small aneurisms appearances may be normal. If the sac has attained to sufficient size it usually appears as a globular swelling in the course of the aorta, generally to the left side, exceptionally to the right. With thin parietes, its pulsations may sometimes be distinctly seen. If it occupies its usual site, near the diaphragm, it may offer unilateral or bilateral obstruction to the movements of that muscle, entailing high thoracic respiration. There may be some turgescence of the superficial veins; the abdomen is otherwise normal. Occasionally icterus is observed, especially so if the aneurism lies in the epigastrium. The general appearance of the patient is indicative of chronic suffering; he is often emaciated, and evinces much mental anxiety. Contrary to what one would expect, edema of the extremities and ascites are not usually accompanying phenomena of aneurism of the aorta.

Palpation: An abdominal aneurism is usually smooth, uniform in feel, globular in outline, immovable, and somewhat compressible. Its characteristic feature is its pulsation; its impulse is systolic in time, and felt both anteriorly and laterally, that is, it is expansile in nature. Occasionally a second impulse or "jogging action" (Walshe), diastolic in arterial time, may be felt. The force of the impulse does not correspond to the size of the tumor; the condition of the contents influences the power of the

pulsation. In 60 per cent. of aneurisms the impulse is felt. It is not altered by change of posture of the patient. A systolic thrill is felt in the majority of cases. The thickness of the sac and the fibrous coagula within may impart to the tumor a deceptive hardness. Though usually globular, the swelling may be quite oblong, and may lie with longest diameter transverse to the axis of the aorta (Hood). The surface of the tumor is generally smooth; surrounding inflammation and adhesions, and irregular coagulation in the sac may make it quite irregular and nodular. Nor is it uniformly elastic or compressible; there may be areas of hardness, with alternate spots soft and fluctuating; Morse found a fluctuating spot through the abdominal wall in the upper part of an otherwise firm tumor. Scheele observed that when the femoral arteries were compressed the pulsations in the tumor became more distinct, pain was increased, and the radial pulse elevated. These observations have been confirmed, yet as the procedure is not essential to a diagnosis, and certainly is attended by the danger of premature rupture, it should not be employed.

Percussion: The tumor, though usually dull upon percussion, may, upon light percussion, be resonant. Inflation of the stomach and bowel will materially aid in showing its relations.

Auscultation: On auscultation there is usually heard a single systolic murmur over the tumor; it is occasionally heard below the tumor. It is most distinct when the patient is in the recumbent posture, and may disappear upon resumption of the erect position. The sound may be harsh and rasping, soft and musical, or blowing, according to the shape of the aortic opening and to the fibrinous deposit. In some instances, a sharp systolic murmur is heard to the left of the lumbar spine, much more marked than in front; it is sometimes heard in the back only. Occasionally a murmur, diastolic in cardiac time, is heard. Leyden's explanation of this diastolic murmur is that the opening of the aneurismal sac into the aorta is narrower than the lumen of the aorta above, hence blood flows in during diastole from the aneurismal opening into the wider space, causing diastolic murmur. Authorities generally consider this diastolic murmur as diagnostic of aneurism; Walshe does not deny its occurrence, yet he has never heard it in aneurism; but he says that it can sometimes be

heard in arteries not diseased, and that it is, therefore, not at all improbable that it does occur in aneurisms. The physical characters of any murmur may change with the growth of the aneurism, and it must be borne in mind that all murmurs are sometimes absent. Auscultation cannot be considered complete that is not made both anteriorly and posteriorly. The murmur heard posteriorly may be double, sharper, more intense than that heard in the precordial region, and may have a booming quality (Dana).

Differential Diagnosis.—In the diagnosis of aneurism of the abdominal aorta we cannot attach great significance to pain; it is a symptom common to many affections, and is sometimes entirely absent in well-developed aneurisms. Neither the presence nor the absence of a murmur is conclusive for or against the presence of aneurism. We are not warranted in proclaiming a positive diagnosis unless there is a "definite tumor which can be grasped and which has an expansile pulsation" (Osler).

Tumors of the pancreas, pylorus, and liver may present many of the features of aneurism, yielding a distinct impulse with each cardiac systole. In the differentiation of such tumors, history, symptoms, and physical signs must all be taken into consideration, and the patient must be examined in different postures. When the patient is placed in the knee-and-chest position, visceral tumors sink away from the aorta and lose their impulse. In other positions, the pulsations of other tumors may be distinctly to and fro, but they never possess that expansile lateral pulsation characteristic of aneurism. The greatest significance may be ascribed to the fixation of an aneurismal tumor; it does not respond to the movements of the diaphragm, and is but very rarely mobile under manipulation.

A pulsating aorta, so frequently seen in neurasthenics and in those suffering from acute anemia from any cause, suggests aneurism to the casual observer; but the absence of any palpable, expansile tumor, and of the usual subjective symptoms of aneurism should exclude this common error.

Prognosis.—A few cases, perhaps, spontaneously recover. The subjective symptoms warrant us in believing that many aneurisms have existed for years before they have been recognized by their physical signs. It is, therefore, difficult to determine their life-history, but their clinical history rarely extends over a longer

period than three years, the subjects dying of some intercurrent malady, or in consequence of pressure-effects, or from rupture of the aneurism.

CONTRAINDICATIONS TO OPERATION.—The surgical treatment of aneurism of the abdominal aorta by any of the several methods suggested is unsatisfactory and disappointing. It must be emphasized that aneurisms situated in the first portion of the aorta, that is, above the renal branches, present the gravest dangers, not the least of which is that the cure of the aneurism may lead to the death of the patient by obliterating the orifices of important visceral arteries (Matas). Cases are not wanting to establish the truth of this conclusion. If the integrity of the sac is destroyed by rupture or erosion, and there is extravasation of blood into the tissues, this condition should preclude the consideration of all operative measures save ligation of the aorta above the aneurism. The constitutional condition of the patient may be responsible for such a vitiated condition of the blood as to greatly lessen its coagulability, an essential to the success of any method. As sacciform aneurisms of anterior evolution are alone amenable to surgical treatment, the difficulties encountered in determining the form of aneurism, in a measure, precludes surgical intervention. An irregular tumor, showing a sac of unequal resistance with diverticula, presents especial dangers; diversion of the blood-pressure to these weak areas may precipitate rupture.

SURGICAL INDICATIONS.—Whatever method of surgical treatment may be contemplated, it should be supplemented by rest, diet, and the administration of iodides.

Ligation of the aorta, which has been done fourteen times to date for various conditions, only thrice, however, for aortic aneurism, by Keen, Milton and Robt. Morris, has been universally fatal. Milton's patient died in twenty-four hours. Morris's case was immediately fatal. In Dr. Keen's case, the aneurism was situated just below the diaphragm, had ruptured, and the extravasated blood had formed an immense hematoma, apparently an utterly hopeless condition, yet the patient lived forty-eight days after ligation, which was evidently completely constricting, as the ligature severed the artery; death was due to rupture of the hematoma into the peritoneal cavity.

It is manifestly difficult, often impossible, even after celiotomy, to determine the site and relations of the aneurismal tumor. It is generally stated that obliteration of the circulation above the mesenteric arteries will inevitably lead to gangrene; Keen's case disproves this; the ligature was applied close to the pillars of the diaphragm; it may have been below the celiac axis, but it "was surely applied above the renal and mesenteric arteries." Brilliant as this achievement was, the results do not justify its repetition.

Pringle refers to five cases, which Hunner thinks were probably all aortic, successfully treated by proximal pressure. If such surprising results were obtained it is difficult to understand why Pringle should limit this method to aneurisms which arise below the aortic bifurcation. The objections to proximal pressure through the parietes are too numerous to require further discussion of it; but, as modified by Keen, after exposure of the aneurism by celiotomy, compression being made with an instrument of special device which lessens but does not entirely obstruct aortic circulation, this method is a suggestion worthy of consideration.

If the sac is small and its intima presumably intact, Macewen's method of needling is especially applicable.

Moore's method, which consists in the introduction of wire or other foreign body into the sac, has been practiced six times for abdominal aneurism, two resulting in cure. This method has been modified by Corradi, by addition of electrolysis. The electrolytic treatment by the Moore-Corradi method is the subject of a most remarkable and excellent contribution by Rudolph Matas, of New Orleans, in which he sets forth its dangers, and restricts it to the comparatively small group of cases in which the aneurisms are confined to the inframesenteric portion of the aorta, and then only after other and safer methods of treatment have been tried. In the 23 cases in which this method has been used for thoracic and abdominal aneurisms, there have been three certain and one probable cure; in nine cases there was decided amelioration of symptoms and prolongation of life. In only six instances was it used in abdominal aneurism, with one cure, Noble's case. The technic of this method has received most careful consideration from Hunner, and while its practice has,

in a measure, the endorsement of Halsted and Finney, it appears to the author that the conclusions of Matas are most tenable.

The hypodermatic injection of a 3 per cent. solution of gelatin in normal saline solution, Lancereaux's method, is often followed by severe reaction, and in one instance (Huchard) by death. It has not received general endorsement. In the nine cases reported by Futcher, there was noted some improvement for a time in seven, but in none was there a cure.

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ANEURISM OF THE BRANCHES OF THE ABDOMINAL AORTA.

RENAL ARTERY.—In the issue of the Philadelphia Medical Journal of May 5, 1900, appears a contribution from Prof. W. W. Keen, in which he reviews minutely the histories of all recorded cases of renal aneurism, and details an additional case of his own. The article exemplifies that author's thoroughness and precision, and it is with pleasure that I accredit him with the points, statistical and diagnostic, given.

Aneurism of the renal artery is of very rare occurrence; 13 cases are recorded; of these 7 were on the right side, and 6 on the left. In 6 cases traumatism is an assigned cause. It may occur without atheromatous changes in the vessel.

Mr. Morris describes two varieties: the first, the small sacciform aneurism; and the second, the false aneurism, in which there is rupture of the artery, the walls being composed of dense fibrous tissue. In Keen's case, the limiting sac was formed chiefly by the pelvis and capsule of the kidney. A false aneurism may be formed by the rupture of a true aneurism, or by the rupture of the artery from temporary occlusion.

True aneurisms, usually small in size, as a rule give rise to no symptoms. A recognizable tumor was present in 5 of the 13 cases—they were all false aneurisms. Pulsation is usually absent; the relatively small size of the artery as compared to the large sac, and the presence of coagula explain this. For the same reasons, thrill and bruit are not to be observed. In true aneurisms there are no urinary symptoms. In cases of rupture, pain, simulating renal calculus, and hematuria have been observed. From this condition the differentiation is made by the discovery of a rapidly growing tumor.

Only 3 cases of renal aneurism have been subjected to operation; all recovered.

HEPATIC ARTERY.—In 1886, Caton collected 11 cases of aneurism of the hepatic artery. The symptoms are very indefinite; in Os-

ler's case they were chiefly those of pyemia. Usually obstructive jaundice is the only symptom; occasionally a small, pulsating tumor may be felt. Schmidt reports death from hemorrhage, due to rupture into the bile-ducts; he finds 5 other such terminations.

SPLENIC ARTERY.—In aneurism of the splenic artery, a few instances of which have been reported, the symptoms have been obstinate vomiting and diarrhea. These symptoms may exist for some time, and finally, when rupture takes place, bloody stools or hematemesis will appear. Hinds and Worthington report an interesting case in a woman 33 years old; ten minutes after a normal labor she had a slight convulsion, was very restless and delirious, and in fifteen minutes was dead; autopsy showed a ruptured aneurism of the splenic artery. In West's case the symptoms of gastric disturbance were present for two months before rupture into the stomach and death from hemorrhage.

MESENTERIC ARTERIES.—Aneurisms of the mesenteric arteries are very rare, usually small, and cannot be diagnosticated *intra vitam*. Several autopsical findings of such pathological curiosities are recorded.

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ANEURISM OF THE ILIAC ARTERIES.

Iliac aneurism belongs rather to the domain of general surgery. The etiology and pathology is that common to aneurism in general. The physical signs are sufficient for a diagnosis.

These aneurisms have become of special interest to the abdominal surgeon since the adoption of a suggestion made by Dennis in 1886, to treat them by transperitoneal ligation. Morton has collected 29 cases treated after this method, with 22 recoveries and 7 deaths; death was due to abdominal complications in only one instance. This technic is now favored by surgeons generally for ligation above the lower portion of the external iliac.

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PRIMARY OMENTAL SOLID TUMORS.

Solid tumors of the omentum occur in the following varieties: *lipoma, fibroma,* and *sarcoma.* Sutton thinks the colloid cancer of the omentum, which is not infrequent and often attains to great size, not primary but a cancerous infiltration from the stomach. I am not able to find an instance of cancer of the omentum, and hence do not include it among the varieties of omental tumors.

Foster reports a case in which a lipoma of the omentum discovered post mortem weighed 55 pounds. Meredith reports the removal of an omental lipoma by enucleation; weight 15 pounds. It consisted of dense lobules of fat. This tumor was elastic, yet evidently solid; the belly was pendulous, and overhung the pubes and groin. It was correctly diagnosed by him upon the following signs: its mobility, its elastic feel, lobular character, and dulness upon percussion. Edebohls cites three cases in which such a growth was diagnosed as fibroid of the uterus.

Fibroid tumors of the omentum are very rare. Clarke, cited by Richardson in Dennis' System of Surgery (who, I may incidentally remark, has written the best article in the English language on Surgery of the Omentum), reports the attempted removal of an extremely large fibroid.

Sarcoma is decidedly the most frequent form of solid tumor of the omentum. Concerning the etiology of these tumors but little is known. Borrmann, after studying the published description of all omental tumors, says "the statement is always found that some portion of the omentum was adherent at some part of the stomach or colon," and he claims that the tumor originally develops from the organ to which it is attached, and that it grows between the layers of the omentum. While this statement may be true in a general way, it does not apply to all cases, and I may be pardoned for introducing here a brief report of a primary sarcoma of the omentum complicated with mesenteric cyst.

The patient was a man forty-five years of age, six feet, seven inches tall, a powerful, raw-boned fellow who had always enjoyed

perfect health until six months before I saw him. He suffered from constipation, colicky pains in the abdomen, lost flesh, and complained of great weakness. Physical examination revealed an abdomen quite distended. The veins were enormously enlarged. Palpation showed an elastic resistance all over the anterior surface of the abdomen. On the left side, by deep pressure, I could get my hand under what appeared to be a tumor two or three inches in thickness. This edge reached from the center of Poupart's ligament to the ribs. I could not move the mass. There was resonance in both flanks. After a few days' observation, a diagnosis of omental tumor was made, and the patient was submitted to operation. A free incision was made from the pubes to above the umbilicus, disclosing a rather unusual and pretty picture. In close apposition to the parietes a mass composed of little lobules of a bright red color filled the entire anterior surface of the abdomen. I can compare it to nothing it so much resembled as a cherry pie. There were no adhesions to the parietes, but it was closely attached to the bladder and pubic region. These being freed, and after extending my incision upward, I was able to raise the entire mass which measured 10 x 14 inches out of the abdomen; I removed it in its entirety by ligating the omentum close to the bowel. Beneath this mass there was a cyst as large as an infant's head which contained a brownish-red fluid. The contents were removed by aspiration, the cyst was then freely opened, packed with gauze, and attached to the edge of the wound. The patient did well for four days, and died suddenly from hemorrhage into the stomach. The autopsy revealed no intraperitoneal hemorrhage, secondary growths, or glandular involvement. The exact nature of the cyst I am unable to state. The stomach was distended with blood, but careful search failed to reveal any gross lesion; the entire mucous membrane showed hemorrhagic infiltration. I had the honor to have associated with me in this case Drs. Buist, Wilson, and Witt, all of Nashville. Microscopic examination of numerous slides showed it to be a myxosarcoma. I am unable to state whether the mesenteric cyst or the omental tumor developed first.

This certainly does not conform to Borrmann's theory of the etiology. There was no stomach- or colonic disease.

PROGNOSIS.—Omental solid tumors may exist for quite a while without giving rise to symptoms, but usually the general condition of the patient evinces disturbance in nutrition. In my own case there was pronounced emaciation, but no other evidence of cachexia. The prognosis is ultimately bad.

SURGICAL INDICATIONS.—Encapsulated growths should be enucleated. Diffuse growths can only be dealt with by amputation of the involved area. The termination of my case shows the danger of the sudden diversion to other viscera of the blood that formerly circulated through a very vascular growth.

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OMENTAL CYSTS.

Cystic tumors of the omentum may be either congenital or acquired. They appear in the following varieties: *serous cysts, blood-cysts, dermoids,* and *hydatids.*

The origin of **serous cysts** is very obscure. They may be inflammatory in nature, may follow traumatism, giving a blood-clot with subsequent liquefaction. Schwarzenberger reports what he calls a lymph-cyst, and says that the origin may be inflammatory. Hearn's case of multilocular cyst of the omentum was undoubtedly congenital. At birth the family physician noticed a distended abdomen sufficiently marked to cause apprehension. This apparently disappeared, and the child lived until six years of age in good health. After that time for two years there was great increase in the growth of the tumor, especially in the last six weeks before consulting Dr. Hearn. Nutrition was fairly good. Operation revealed that it was a typical multilocular cyst, and the history warrants the conclusion that it was congenital. The patient made a rapid recovery. It must be remembered that beyond the sense of weight in the abdomen there were practically no symptoms.

Gooding's case was a typical serous cyst of traumatic origin.

The existence of **dermoid tumors** in the omentum has been denied, and it is true that there is no authenticated case that we can find; yet embryonic inclusion should produce such a tumor here as well as in the mesentery, of which there are several indisputable cases.

Solitary **echinococcus cysts** are of very great rarity, yet Witzel gives a very interesting account and detailed study of a case in a girl ten years of age, from whom he removed hydatids of the omentum by laparotomy, fixation and drainage of the sac.

Cysts of the omentum usually lie at or below the umbilicus, and produce a peculiarly pendant abdomen. The distention of the abdomen may be enormous, and this feature is misleading, as it diverts attention from the omentum. By palpation the tumor is usually round, smooth, fluctuant; but multilocular cysts

are lobulated. Unless adherent to viscera or parietes, they move synchronously with the diaphragm. They permit of free movement laterally and upward, but not downward. They are dull upon percussion, with surrounding resonance. By their weight and size they may cause much discomfort. In Witzel's case of hydatids there was severe pain, paroxysmal in character, which he ascribed to torsion and traction.

Omental cysts may be mistaken for encysted peritonitis. Here the history, the possible tuberculous origin, the fixity of the tumor, should enable us to distinguish it.

In ovarian tumors we find many signs in common. The progress in the growth of ovarian tumors from below upward, while in omental cysts the reverse is found, is of valuable diagnostic significance when obtainable. Ovarian tumors which lie out of the pelvis and in the abdominal cavity necessarily draw the uterus upward, so that by vaginal examination we shall find the uterus high in the pelvis, and usually, as is shown by bimanual palpation and the sound, laterally displaced. Omental cysts, on the other hand, as they descend will force the uterus downward either in ante- or retroflexion. Ovarian tumors may be moved laterally and downward when small and pedunculated, but they cannot be moved upward to any extent. This upward movement is very characteristic of omental tumors. Ovarian tumors are not influenced by respiration; ovarian tumors are dull from the pubes up, except in very rare instances.

Movable kidney, especially when hydronephrotic, offers diagnostic confusion; but a study of the history and symptoms in each, together with the colonic resonance on the inner side in renal tumors, should enable us to differentiate them.

In themselves omental cysts are harmless, but their size and pressure, and inflammatory complications may so impair nutrition that the patient will die of exhaustion.

These cysts should be removed in their entirety, with ablation of the involved portion of the omentum.

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TORSION OF THE OMENTUM.

Dr. Joseph Wiener in a contribution to the Annals of Surgery, Vol. XXXII, 1900, entitled "A Study of Intra-Abdominal Omental Torsion," gives us practically all that is known concerning this very rare condition. He collects seven cases, including his one, to which I may add Eitel's case reported in the New York Medical Record, May 20, 1899, which was somewhat different in pathology, yet produced the same symptoms. From Wiener's study the following conclusions may be drawn: Omental torsion occurs from fixation of the distal portion of the omentum, usually in a hernial sac; forcible efforts at reduction may return part of the omentum into the peritoneal cavity, producing torsion and twisting of the pedicle; or the peristaltic movement of the bowel itself may cause rotation of the omentum and strangulation; the accident is far more common in males than in females; when torsion takes place in connection with hernia, the diagnosis is usually that of incarcerated hernia. Peck reported to the New York Surgical Society a case in which the omentum was attached to the colon, producing sudden onset of symptoms resembling obstruction.

In all the cases the urgency of the symptoms was recognized. In Eitel's case a large abdominal tumor was produced. At operation the omentum was found tightly rolled upon itself, constricting the veins and giving rise to ascites, for which the patient had been repeatedly tapped. The omentum was simply unrolled and the symptoms relieved. The immediate cause in this case was traumatic; the patient had carried a heavy box, which was pressed upward and against the abdomen.

PRIMARY RETROPERITONEAL AND MESENTERIC SOLID TUMORS.

DEFINITION.—The term implies a solid neoplasm growing behind the peritoneum or between its folds, and not generally connected with any of the retroperitoneal organs.

FREQUENCY.—There seems to be an impression among surgeons that these tumors are so rare that they are scarcely worth consideration, and this is borne out somewhat by Mr. Lockwood's statement that no specimen of retroperitoneal sarcoma, which is the most frequent form of tumor, had been presented to the Pathological or Medical Society of London prior to 1895. In a recent discussion in the Medical and Chirurgical Society of London, following a report of a case by Mr. Sheild, it appears that the members were quite unfamiliar with the subject. The author read a paper before a body of America's leading surgeons, and none present had had any operative experience with these tumors, yet numerous cases are recorded, especially within the last few years.

VARIETIES.—The types of retroperitoneal tumors belong to the connective-tissue group. The innocent tumors are lipoma, myxoma, and fibroma; the malignant is sarcoma. In analysis of twenty cases of solid retroperitoneal tumors collected by Rogowski, sarcomatous tissue was found in every one.

ETIOLOGY.—There is nothing definitely known as to the cause of these tumors; but from instances quoted above and others found in the literature it seems that sarcomatous degeneration is engrafted upon preëxisting innocent growths, whose presence has been recognized for many years. There is one instance in which preëxisting retroperitoneal, possibly subdiaphragmatic, abscess may have had some causal relation.

Age: They usually occur after maturity, between the ages of thirty and fifty years. Primary sarcoma is very rare in children, yet Arnstein collected ten cases, including one of his own, a female four years old.

Sex: It occurs with equal frequency in both sexes.

PATHOLOGY.—**Origin:** The structures involved primarily show that these growths originate in the retroperitoneal cellular tissue, retroperitoneal and mesenteric glands, from the sheaths of the great blood-vessels, and from the vertebræ. In one inoperable case of fibrosarcoma upon which I made an autopsy, the tumor appeared to arise from the root of the mesentery, its other attachments being very slight. In one of my cases the tumor sprang from the sacrum; in the other, a large myxosarcoma, the origin seemed to be from the cellular tissue of the loin.

Sarcoma: As above stated, sarcoma is the predominating tumor. The microscopical picture is generally of the spindle-celled sarcoma, yet in literature we meet with such descriptive terms as myxosarcoma, liposarcoma, lymphosarcoma, and cystic sarcoma. The last variety is so closely allied to cysts that Moynihan classifies it as one of the varieties of mesenteric cysts.

An important pathological characteristic of retroperitoneal sarcomata is that they are encapsulated, yet metastases occur in about one-half the cases; liver, lungs, and mesenteric glands are the parts involved. These tumors sometimes attain to an enormous size. In Waldeyer's case, quoted by Rogowski, the sarcoma was joined by a large fatty tumor. Other cases are reported of sarcoma associated with other growths.

Lipoma: The most characteristic specimen of lipoma was obtained at autopsy by the late Prof. Mudd, of St. Louis; the tumor weighed 66 pounds, and had a distinct fibrous capsule.

Fibroma: I removed post mortem a tumor that had existed fifteen years, not connected with any of the viscera, which was distinctly encapsulated and attached by a strong broad pedicle at the root of the mesentery. It was declared by the microscopist to be a myofibroma.

SYMPTOMS.—Retroperitoneal tumors possess certain well-marked characteristics not found in primary intra-abdominal neoplasms. The latter originate primarily between the duplications of the peritoneum and are attached to the organ from which they spring; while retroperitoneal tumors originate in the retroperitoneal space, and as they develop they encroach upon the peritoneal cavity, pushing the organs contained therein either forward or to the side in a most typical manner (Witzel).

A patient bearing an innocent retroperitoneal tumor of moderate size may evince no constitutional disturbance; but usually the large size of the tumor causes disturbance of the digestion and respiration. The kidneys are frequently displaced and compressed by the growth. The blood-vessels and lymphatics are obstructed. The intestines may be occluded by pressure. Consequently, general emaciation is one of the most constant and oftentimes the only symptom, barring the presence of the tumor, of innocent neoplasms. But unfortunately, as we have shown, these tumors are often primarily malignant, as is evidenced by the rapidity of their growth and the early and pronounced cachexia.

Pain, when present, is usually due to constipation, with threatened obstruction, and may be associated with nausea and vomiting.

Tenderness is absent unless there is peritoneal involvement.

In many instances, from pressure, metastatic glandular involvement, and peritoneal irritation we find ascitic fluid, often so great as to obscure the physical signs. A valuable symptom, and one which not infrequently occurs early, is edema of the extremities. As before stated, the renal secretion is often disordered, but in only one instance has blood been found in the urine.

PHYSICAL SIGNS.—**Inspection:** The appearance of the abdomen in these growths is by no means characteristic. They grow anteriorly, as Vander Veer says, in the direction of least resistance. The greatest swelling is usually in the upper portion, at or above the umbilicus. When of large size they may produce a very pendulous belly (Mudd's case). The abdominal distention is usually asymmetrical. The size and location of the tumor, therefore, influence the morphology. The superficial veins are very much enlarged, and respiratory movements of the tumor may be observed. In Ransohoff's case the tumor lay chiefly in the right side, and moved distinctly with respiration. Especially in mesenteric tumors is the function of the intestines impaired; this obstructive feature, being chronic in character, develops intestinal hypertrophy, and I have observed distinct peristaltic patterns in one case.

Palpation: Tumors mesenteric in situation are exceedingly movable, while those close to the parietes are fixed. They are

usually smooth, their borders distinctly defined. They are firm in some instances, elastic but not fluctuant unless undergoing cystic degeneration. In Mudd's, Ransohoff's, and Guillemain's cases of large retroperitoneal lipomata, fluctuation was apparently distinct. Lipomata impart a sensation more like that of the liver, and they are often lobulated. Palpation and conjoined manipulation will negative their association with the pelvic and other organs.

Percussion: This varies with the size and position of the tumor. That this sign may be employed to the full of its diagnostic value, the bowels should be freely evacuated, and the patient should be examined after colonic inflation with gas, and in different positions. Auscultatory percussion is of infinite value in determining intestinal relations. Assuming the case to be one in which the tumor is of moderate size, say as large as a child's head, if central in position it is surrounded by a zone of resonance; if lateral in position, the resonance is at the umbilical border. But one essential and characteristic feature of these retroperitoneal tumors is that there is fixed linear resonance, elicited by light percussion, either upon one side or across the surface of the tumor. As Witzel says, in these retroperitoneal tumors the colon bears a typical and most diagnostically important relation. We must remember that these tumors originate near the spine; therefore, when small, the colon may be to the outer side. As they grow the colon lies in front; and when they attain to great dimensions the colon lies across or to the umbilical border. In small mesenteric growths the overlying intestines may give central resonance; it is in this form of tumor that a change of position will give us varying resonance. The accompanying ascites, which is not an infrequent complication, will often obscure the presence of the tumor, or mislead us by varying dulness.

One of the most important of the diagnostic signs, revealed both by percussion and palpation, is that the tumor is separable from the solid viscera; yet in tumors of large size with right lateral evolution the colon, ascending and hepatic, is displaced downward and to the center, and there is no differential resonance between the liver and the tumor. If the patient is examined in Trendelenburg posture, an area of resonance in the lower

zone of the abdomen, skirting the pubis and Poupart's ligament, can usually be outlined. This is of immense value in eliminating uterine and ovarian growths.

COMPLICATIONS.—The most usual complications occurring in the course of solid mesenteric tumors are ascites and intestinal obstruction.

DIFFERENTIAL DIAGNOSIS.—Innocent growths are recognized by their long duration and the comparative absence of symptoms; and fatty tumors, especially, by their lobulated character, elastic, almost fluctuant feel.

The malignant tumors are rapid in growth, produce complications of ascites and edema, and are quickly productive of cachexia.

Tumors of the solid viscera, spleen, kidneys, and liver, may be mistaken for these growths; yet the functional and constitutional disturbances, and carefully applied physical tests, should make the differentiation. In August last I removed a retroperitoneal myxosarcoma, weighing 16½ pounds. The only marked symptoms were periodic attacks of cramping pain, vomiting, and constipation. The physical signs led to the erroneous diagnosis of renal tumor.

PROGNOSIS.—The prognosis in lipoma and other forms of innocent tumors is ultimately as grave as in sarcoma, the disturbances from pressure finally bringing about death from marasmus. And, as we have shown, the possibility of change to malignancy must be taken into consideration.

As illustrative of operative results we would refer especially to the successful cases of Harris and Herzog, who removed a large lymphsarcoma and with it 51 cm. of attached intestine. The patient recovered. Also the case of Shepherd, of a fibromyxoma weighing 13 pounds, with resection of 7 ft. 8 in. of intestine with recovery and restoration of his patient to health.

CONTRAINDICATIONS TO OPERATION.—Pronounced cachexia, metastasis, and manifest universal attachments are contraindications that interdict even exploratory incision. After opening the abdomen, which procedure in every case is more or less exploratory, the relations and attachments of the tumor may contraindicate further effort.

SURGICAL INDICATIONS.—The known encapsulated character of retroperitoneal sarcomata, and oftentimes their long duration without metastasis or cachexia, encourage the surgeon to undertake operation. The dangers presented are hemorrhage, and necrosis of the intestines or parietal peritoneum.

The surgical procedure is enucleation of the tumor, but in doing this we must bear in mind that the intestinal circulation may be cut off. When its integrity is impaired, resection of the gut, as in the cases above quoted, is necessary. Drainage must be provided, usually best obtained through the loins.

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RETROPERITONEAL AND MESENTERIC CYSTS.

DEFINITION.—**Retroperitoneal cysts** are fluid or semi-fluid neoplasms, originating and residing entirely behind the peritoneum lining the posterior abdominal parietes.

Mesenteric cysts are those fluid tumors that originate and grow in structures lying between the mesenteric, mesocolonic, and mesorectal folds. Retroperitoneal cysts by their growth and extension may separate the mesenteric folds, so that it is often impossible to determine to which variety these cysts belong; and as they present many features in common, we shall consider them as one.

FREQUENCY.—Augagneur, in 1886, found recorded only 19 mesenteric cysts. Moynihan, in 1897, was able to gather 113 cases. The latest statistics are those of Dowd, who makes reference to 145 cases. Mr. Tait in his 2,000 laparotomies did not meet with this condition. Spencer Wells found only two cases. The condition is scarcely alluded to in text-books.

VARIETIES.—Attempts to classify these cysts have been made upon different bases. Arranged according to the nature of their contents we have (1) *serous cysts,* (2) *chyle-cysts,* (3) *hydatid cysts,* (4) *blood-cysts,* (5) *dermoid cysts.* Malignant tumors undergoing cystic degeneration are not properly cysts, and hence are not included. This classification based upon the contents of the cyst is open to criticism. As yet our knowledge of the histological structure of the cyst-wall is not sufficient to warrant us in attempting a genetic classification.

ETIOLOGY.—Przewoski, quoted by Obalinski, regards *serous cysts* as embryonic in nature, first, because they originate in the immediate vicinity of the kidney, where certain metamorphoses may take place in the remnants of the Wolffian bodies and ducts of Müller; and secondly, from their contents.

Blood-cysts, mesenteric and retroperitoneal, may be due to traumatism, and non-absorption of the effusion (Schönwerth's case); or to hemorrhage into a preformed cyst, as observed by myself.

Chylous cysts have the following origins: (1) thoracic duct, (2) receptaculum chyli, (3) lacuna mesenterica, (4) mesenteric glands, (5) rupture of a chyle-duct between the folds of the mesentery. Küster thinks that there is no doubt whatever that many of these cysts are congenital, and are caused by fetal inclusion. Rasch says that there is nothing known as to their etiology. Gusserow thinks the most common origin is in degenerated mesenteric lymphatic glands. Virchow ascribes them to the same origin. According to Carson, in eleven cases that he analyzed, three were due to dilatation of the lymphatics and eight to degenerated glands.

Dermoids, wherever found, according to Cohnheim, are due to the inclusion of the ectoderm; therefore, these tumors are always congenital in origin. Several instances of mesenteric dermoids are reported.

Hydatids are due to the lodgment of the embryo of the tenia echinococcus in the subperitoneal spaces, where it is transformed into a cyst, the condensation of the tissues forming an enveloping capsule.

PATHOLOGY.—Cysts are pathologically classified according to their contents, the histological structures of the cyst-wall, and their location.

Mesenteric Serous Cysts: In regard to the development of mesenteric cysts, it must be recognized that we have an intraperitoneal serous cyst which may be mistaken for a mesenteric one. Within the peritoneum there are three special fossæ, the subcecal-iliac fossa, the plica duodenojejunalis, and the intersigmoid fossa; these cavities in the peritoneum in embryonal development may become very much deepened, and often the seat of hernia. If these fossæ become inflamed from any cause whatever, they may be entirely or partially obliterated. If obliterated at the neck, as is often seen in inguinal and femoral herniæ, we have a serous sac in which there may be a large accumulation of fluid. Such sacs have an inner lining of endothelium, by which we may distinguish them. Heinrich's case was of this special variety. The condition of abnormally large fossæ is congenital, but its exclusion and the effusion which takes place into it is a pathological process.

Some true mesenteric cysts may find their origin and have their pathological characteristics like the above. In Dowd's case, which he so thoroughly studied, we find that the main cyst-wall was composed of dense fibrous tissue; on its inner surface there were indistinct small cells irregularly arranged, apparently the compressed remnants of the epithelial layer. Küster, quoted by Heinrich, found flattened epithelium in a mesenteric cyst. McDonald found cylindrical-celled epithelium, and Thornton found granular cells with large nuclei, such as are met with in ovarian cysts. All these observers were struck by the similarity in structure of the cyst-walls to those of ovarian cysts. The pathologist declared that Dowd's specimen could be used for class-room demonstration of ovarian cystic adenoma, nor in this particular instance did the resemblance cease here. The specific gravity of the fluid was 1010, alkaline, and somewhat viscid. Microscopically, there were blood-corpuscles, cholesterin, and large granular cells. But more significant than all these was the presence of pseudomucin, which is a constant element in the fluid of glandular ovarian tumors. Such findings led Dowd, and before him Przewoski, to conclude that many of these cysts were of embryonal origin.

Not presuming to dispute this statement, I think it reasonable to assume that the closing off of any of the peritoneal fossæ could give us an intraperitoneal cyst, which in its development would appear as mesenteric or entirely retroperitoneal. Dowd makes a great point in his argument of the location of his cyst, which was above the colon; it could have developed from the duodenojejunal fossa.

Retroperitoneal serous cysts may originate from traumatism, the condensation of the tissues forming the fibrous cyst-wall, composed entirely of connective tissue; or from the Wolffian body, or the ducts of Müller.

Preformed cysts may have hemorrhagic effusion into them, obscuring the character of their original contents.

Chylous cysts have variable, at least somewhat doubtful, contents. Remembering their various points of origin—it may be chyle from receptaculum chyli, or thoracic duct, or, as we have stated, most frequently from a degenerated gland—we find their contents to be either pure chyle of milky or creamy color and of

that consistency, or very thin, or pulpy. It does not coagulate on standing. The varying colors are due somewhat to the admixture with blood. Microscopically we find fatty globules and granular corpuscles, without any other morphological mixture.

The nature of the cyst-wall is peculiar and interesting. It is composed entirely of connective tissue, and there is an absence of endothelial and epithelial cells.

The contents of dermoids consist of fatty matter, flattened epithelial cells, and dermal elements. As previously stated, the origin of these dermoids, while obscure, may be explained by sequestration of the entoderm.

Hydatids: The contents of a hydatid is clear fluid, slightly opalescent, alkaline in reaction, sp. gr. about 1010. Scolices are present usually, and when found are diagnostic. The cyst-wall is composed of the condensed connective tissue.

The typical retroperitoneal cyst is usually fixed, confined by the tightly overdrawn peritoneum. When of traumatic origin they may be associated with intraperitoneal adhesions. Mesenteric cysts naturally enjoy greater mobility. Blood- and hydatid cysts are especially prone to degeneration and abscess-formation. Rupture from overdistention or accident may occur in any form of these cysts, giving rise to serious intra-abdominal complications. Occasionally only do we find intestinal obstruction as a complication of cystic tumors.

Symptoms.—The diagnosis of mesenteric and retroperitoneal cysts is fraught with the greatest difficulty. Schede denies the possibility of diagnosis. Löhlein states that exploratory laparotomy is the only means. When these opinions were expressed there were 90 cases on record. Perhaps double that number are now reported, and within the last ten years the diagnosis has been repeatedly made. It is true, we have not reached that refinement of diagnosis to enable us to individualize the different varieties, but, as we hope to show, the surgical diagnosis of mesenteric and retroperitoneal cysts can be arrived at by exclusion and by the physical signs they present.

Physical Signs.—**Inspection:** Mesenteric cysts are usually central in location, or lie to the right side. When they attain any marked dimensions, as large as a cocoanut, they generally lie close to the umbilicus. Mesocolonic cysts occupy a lateral

position unless of large size. Changes in the position of the tumor may be observed at different examinations. When the abdomen is greatly distended, the superficial veins may appear enlarged.

Serous mesenteric and retroperitoneal cysts are frequently multilocular, and hence give an asymmetrical appearance to the abdomen.

Palpation: These tumors are usually smooth, occasionally lobulated, borders distinctly defined, though the sac has been found so flaccid that it has simulated ascites (Heinrich's case); soft, elastic and markedly fluctuant. Unless fixed by adhesions, or typically retroperitoneal they are freely movable. They do not move with respiration. The borders of the solid viscera may be felt distinct from the tumor. When large they lie in close apposition to the walls of the abdomen. Small mesenteric cysts are characterized by their exceeding mobility, which exceeds that of movable kidney.

Percussion: The signs disclosed by percussion depend largely upon the size of the tumor. If the tumor is so large as to greatly distend the abdomen, general dulness is all that is obtained, but a tumor of lesser dimensions yields upon percussion significant information. It is surrounded by an area of resonance; this is valuable in excluding the spleen and liver above, and uterine and ovarian tumors below. The dulness may be absolute when the tumor rests in apposition to the abdominal wall. Lineal tympany is found upon light percussion either in front of or to the sides of the tumor; this is fixed and not altered by any change in the position of the tumor; it is the attached coil of intestine. In small mesenteric cysts the intestines may so overlie it that dulness cannot be obtained.

In mesocolonic cysts inflation of the bowel greatly assists in defining their position by developing linear tympany. Mesocolonic cysts, like renal tumors, tend to push the colon inward, so that resonance is only found on their umbilical border.

In especially large cysts that reach to the pubes, examination in Trendelenburg posture may cause the tumor to gravitate and develop hypogastric tympany, a valuable sign.

Auscultation: Negative.

Exploratory Puncture: This has been done repeatedly without ill effects, yet I oppose it upon general surgical principles. And, furthermore, it is needless; the application of the ordinary methods of diagnosis have determined the surgical indication, if not the exact pathological condition.

DIFFERENTIAL DIAGNOSIS.—**Tumors of the Intestines:** The mobility of the tumor and its ordinarily central position would suggest its intestinal nature. Intestinal tumors are usually accompanied by symptoms of obstruction, partial or complete; paroxysmal pain, attacks of constipation with alternating attacks of relieving diarrhea, vomiting, and peristaltic patterns—all of which symptoms are absent in mesenteric cysts.

Distended Gall-Bladder: Simple and auscultatory percussion will exclude liver and spleen, yet we must bear in mind that a distended gall-bladder may occupy very unlooked for positions, may have linear resonance owing to adhesion of small intestine, may even be separated from the liver by an area of resonance; but in this we have a history of gall-stone colic, and the general phenomena of obstructive jaundice.

Ovarian cysts are excluded by their intrapelvic origin and growth upward, their intimate connection with the uterus, by the dulness which exists from the symphysis and Poupart's ligament upward to the crown and flank resonance. Mesenteric cysts originate above and grow downward, have no relation to the uterus and appendages, and there is resonance between the tumor and the pubis.

Movable kidney is differentiated by its shape, by its tendency to assume its normal position when the patient is recumbent, by the occurrence of Dietl's crises, by its far greater frequency in neurasthenic women, and by its association with general enteroptosis.

PROGNOSIS.—Rapidly growing mesenteric cysts cause death from exhaustion and interference with digestion. This is especially true of chylous cysts. The liability to rupture or to peritoneal inflammation renders them dangerous. Occasionally intestinal obstruction occurs, either by the gradual occlusion of the gut by pressure, or by constriction and torsion from pedunculated cysts Large growths interfere with the general functions.

Begonin says that cystic tumors were cured in 88.93 per cent. by simple incision and drainage in 49 cases. Extirpation of the tumor gives a mortality rate of 52 per cent. Postoperative mortality is due either to shock, hemorrhage, or intestinal gangrene.

SURGICAL INDICATIONS.—Ordinary serous retroperitoneal cysts, when small, may be enucleated; if their attachments to the intestines are too great the sac should be attached to the parietal wound, drained, and packed with gauze.

Chylous cysts should invariably be drained; no effort should be made to excise them. In fact, every case must be a law unto itself. The integrity of the intestinal circulation must be preserved, and it is, therefore, wise to drain rather than to resort to resection of the intestine.

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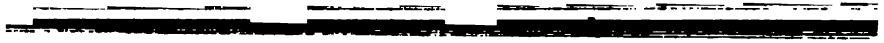

VESICO-UMBILICAL FISTULA.

DEFINITION.—A patulous urachus communicating with the bladder below and opening upon the skin-surface at the umbilicus above.

SYNONYM.—Urachal fistula.

Urachal fistulæ may be complete or incomplete. The incomplete variety has a non-patulous umbilicus, but the dilated canal opens into the bladder; Mr. Stanley had such a case, as recorded by Doran. Should the canal remain patulous throughout its extent from bladder to umbilicus, we have a true urachal fistula; Jordan Lloyd refers to several such cases in children. It is unusual to find this condition existing in adult life; a striking case is recorded by Freer, of Washington. Such fistulæ in children may spontaneously close, but many of them have been known to reopen in after-life; Vander Veer's case so behaved.

The phenomenon of the escape of urine into the urachal canal when there is obstruction to the exit of urine from the urethra seems to imply that there is a true valvular fold, as described by Wutz, at the vesical orifice of the urachal canal.

A urachal fistula is recognized by the escape of urine from the umbilicus. We are frequently able to pass a sound through the fistula into the bladder; or if the bladder is distended with fluid on account of obstruction to the outflow of urine, pressure over the bladder will force the fluid along the urachus to the umbilical opening. Those cases that are not congenital are usually associated with some vesical or urethral obstruction.

In the management of urachal fistulæ there are three indications: first, to dissect out the urachus; second, to close the vesical communication with Lembert sutures; third, to remove any obstruction to the urinary outflow, if such be found.

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CYSTS OF THE URACHUS.

DEFINITION.—An accumulation of fluid in an abnormally patent urachus, lying extraperitoneally in the anterior abdominal parietes.

FREQUENCY.—Judging from text-book literature on the subject, one would consider cyst of the urachus to be a rare affection, and the casual reader would be inclined to think that to Mr. Tait alone had been vouchsafed the privilege of observing this phenomenon. While neither of these propositions is entirely true, it certainly appears upon evidence that Mr. Tait alone enjoyed the distinction of being able to diagnose this condition. Luschka thus wrote in 1862: "I have no doubt of the occurrence of large cysts requiring surgical interference, and that these cystic swellings found in the abdominal wall have their origin in the development of the urachus." The report of Hoffmann's four cases, in 1870, established the fact that cyst of the urachus was a definite pathological condition, amenable to surgical relief. However, one of Hoffman's cases, which contained fifty liters of fluid, is rejected by Wutz and by Doran, who consider it merely a case of encysted peritonitis. Then followed the inaugural address of C. Christian Wolff, in 1873, with a full account of a carefully noted case upon which he operated, with suggestions as to diagnosis. In these articles we have about all the information obtainable until the article by Mr. Tait appeared, in 1883; then his series of twelve cases reported to the British Gynecological Association, in 1886. He has since met with several others, four of which were recounted by Robinson. Yet I am pleased to remark that the studies of three Americans, F. Byron Robinson, James A. Freer, and Edward J. Ill, have thoroughly systematized the subject, elucidated its pathology, simplified the diagnosis, and suggested rational treatment. Mr. Doran, I am pleased to say, considered my contribution valuable.

ETIOLOGY.—Simultaneously with the development of the amnion is the appearance of the allantois. This structure takes its origin

at the lower wall of the cloaca, is at first solid, later becomes spherical and hollow. As it advances forward toward the periphery of the ovum it becomes wider and longer, reaching that part of the endometrium where it takes part in the formation of the vascular layer of the chorion, conducting the two arteriæ umbilicales, establishing the fetal and maternal circulation.

That portion of the allantoic vesicle remaining within the body-cavity is generally spoken of as the urachus, a portion of which is destined to form the urethra and bladder, which are the "permanent functional parts." The remainder forms generally an impervious cord stretching from the summit of the bladder to the umbilicus; this is the urachus proper. Luschka declares that in the majority of males the urachus is found to be partially open and lined with mucous membrane; and it is remarkable that it is covered with flattened epithelium, when we remember that primitively it coalesced with the intestinal canal, which has cylindrical epithelium.

Should there be ever so small communication with the bladder, inflammation of that organ or interference with its function may force the urine into the urachus and rapidly develop a urachal cyst. Dr. F. Bramam, of Berlin, closes the report of a case with these instructive remarks: "The etiology of this trouble is the chronic cystitis from which the child suffered, causing difficult evacuation of urine from the bladder. The repeated stasis of urine resulted in the hypertrophy of the bladder, and the convulsive closure of the urethra which occurred from time to time during the four years of her ailment caused the urine to be forced into the urachus, which it dilated and eventually perforated."

Age: These cysts may be met with at any age, but they are far more common in middle life. The extremes are five and sixty-six years. In twenty-five cases in which the age is given there were only five cases under seventeen years, one female and four males.

Sex: Sex is an element for our consideration. Of thirty-seven cases collected from various sources, in which there was a distinct abdominal tumor, twenty-nine were females, only eight were males. On the other hand, vesico-umbilical fistulæ are far more frequent in males.

PATHOLOGY.—Luschka describes the minute anatomy of the urachus as follows: (a) *A structureless basement membrane.* This is a very delicate, transparent, structureless membrane not acted on by acetic acid. It can be separated into smaller fragments, which have a tendency to arrange themselves into folds. (b) *A fibrous layer.* This is attached to the outer side of the basement membrane and distinctly separated from the neighboring cellular tissue. It is interspersed with various long nuclei, having a dark contour, irregularly scattered through the fibrillary interstitial substance. (c) *Epithelium.* The epithelial layer is found on the inner side of the basement membrane and consists of a variety of cells, such as are found in the bladder, ureter, calices and pelvis of the kidney. They are either ovoid or polygonal nucleated lamellæ, and are of variable form and size.

Either extremity or any part of the tube may remain open, constituting a variety of abnormalities presenting interesting pathological phenomena. Abdominal surgeons have frequently encountered small sacculated dilatations of the tube. Ill describes it as "presenting the appearance as if it were knotted or had excrescences." It has been better likened to a string of small sausages. These small cystic expansions of the urachus, if they escape injury or infection through the umbilicus or bladder, may exist throughout life unobserved. A true allantoic or urachal cyst sufficiently large to be of importance usually arises from the dilatation of the greater part of the urachus. The vesical and umbilical ends being occluded, such cysts very properly belong to the genus designated by Bland Sutton as "tubulocysts." A cystic expansion of the vesical end of the urachus communicating with the bladder is not a true cyst, but, as designated by Schullenbach, a cystic ectasis. An excellent illustration of this variety may be found accompanying the article of J. B. Wutz. Byron Robinson also gives a very good cut of this variety of urachal cysts, removed post mortem from a boy ten years old.

SYMPTOMS.—Usually there exists in an acute or chronic form some pronounced disease of urethra or bladder in which has occurred retention of urine or great vesical tenesmus. The history of these cases is that of acute illness with rather sudden development of abdominal tumors. This statement is at variance with

that of Dr. Ill and others, yet a careful study of reported cases justifies it. The symptoms are rather constant. The patient feels ill, loses flesh, and in many cases looks distinctly tuberculous. Pain is a conspicuous feature; it is sometimes intense and paroxysmal, but usually described as dull and heavy, due to tension and pressure in the pelvis and lower abdomen. Nausea and vomiting are frequently observed; extreme prostration, faintness, sickening sensations, are nervous symptoms of note. There is usually constipation. Urination is generally frequent, but there is marked diminution in the amount of urine, which is high-colored and not infrequently contains mucus and pus. And there is often dysuria; the only case in which I can find this symptom absent was one carefully recorded by Freer.

Physical Signs.—The physical examination reveals an abdominal swelling lying chiefly below the umbilicus. If small and not inflamed, with relaxed abdominal walls, it is easy to demonstrate its mural character. If the tumor is of large size the appearance of the abdomen is peculiar. The distention is chiefly below the umbilicus, frequently asymmetrical. It is quite flat upon the upper surface, bulging laterally. It is immovable, the parietes inseparably connected with the cyst. It is uniformly soft and fluctuant. The percussion-test, our chief reliance in reaching a diagnosis, is peculiar and characteristic in well-marked cases, and as this physical sign has been so well described by Mr. Tait, I quote his words: "The pelvic dulness is absolute, whilst the dulness which is contained above the umbilicus is not so, although it is perfectly certain that the wave of fluctuation passes through one volume of fluid not intercepted by any cyst-wall." He explains the sign by a peculiar reflection of the peritoneum. The intestines which normally occupy the pelvis are forced upward and underlie only the upper part of the cyst. To briefly describe the percussion-sound, I would say that it was *fading dulness* from below to the upper limit of the tumor.

Bimanual examination usually shows a retrodisplaced uterus, very low in the pelvis, entirely separate from the tumor. If the tumor occupies the pelvis the entire vaginal vault will be encroached upon by a soft elastic mass. Marked displacement and distortion of the bladder may be discovered with catheter and cystoscope.

Polygonal epithelium discovered in the aspirated fluid establishes beyond question the diagnosis. It may be well to remark here that the contents of urachal cysts are sometimes surprising. The bright yellow color of the fluid puzzled Mr. Bantock. Bramam, of Berlin, encountered four large gall-stones, Rokitansky found thirty-one.

All cases of urachal cysts do not present the pronounced clinical features above given. Many behave very innocently, occasioning only moderate distress until they attain quite a growth. Such was the history of my case.

DIFFERENTIAL DIAGNOSIS.—The most pardonable error in diagnosis is to mistake a urachal cyst for a parovarian cyst. The condition has been often diagnosed as encysted tuberculous peritonitis, gastritis, hydronephrosis, and the true condition disclosed only at operation. I cannot refrain from calling attention to the marked parallelism existing between the symptoms of this condition and of ectopic gestation. The great prostration, the rapid pulse, the recurring syncope, the nausea, the paroxysmal pelvic pain, menstrual disorder, and vesical irritation are symptoms common to both. The passage of decidua and thorough bimanual examination will alone make the differentiation in some cases. Roser speaks of a case of cyst of the omphalomesenteric duct which he had mistaken for cyst of the urachus; Fischer, of Breslau, reports a similar operation.

PROGNOSIS.—Conservatively treated by incision and drainage, the prognosis is excellent in these cases.

SURGICAL INDICATIONS.—Incision into the cyst, thorough evacuation of its contents, irrigation of the cyst-cavity, mopping out with a solution of iodine, and drainage should be the chosen method of treatment; there is no doubt that there are many of these cysts which dip down into the pelvis that cannot be removed, and we are forced to adopt as a *dernier ressort* something like the above plan of treatment.

The more radical operation of enucleation of the sac, when it is possible, has a finish, a completeness about it attractive to the surgeon. When the cyst has been dissected away from a large area of peritoneum, it is best to trim the detached serous membrane, even if it appear unhurt. In my case death was undoubtedly due to sloughing of the detached peritoneum. Care should

be observed to ascertain the relations of the bladder to the cyst; and after the evacuation or removal of the cyst, the bladder should be distended in order to expose any urinary fistula, which, if present, should be closed.

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FIBROMYOMA OF THE UTERUS.

DEFINITION.—A tumor of the uterus composed of fibrous and unstriped muscular tissue in varying proportions, the muscular element usually predominating. Occasionally the formation is diffuse, but is generally exhibited as single or multiple nodular masses.

SYNONYMS.—The terms myoma, fibroma, and fibroid tumor will be used interchangeably.

FREQUENCY.—2.8 per cent. of 30,000 patients visiting Chrobak's gynecological clinic had myomatous uteri. Of 9,227 females with neoplasms, in 883 the growth was uterine myoma (Williams). Such statistics give a very inadequate idea of the frequency of myoma. They do not take into the estimate those patients in which the tumor has a symptomless existence. Bayle says that 20 per cent. of all women over thirty-five years of age bear myomata. Homans asserts that in fifteen years' practice he has met with something over 500 cases of myoma of the uterus. In my college clinic, the patients attending being chiefly negroes, I find, after displacements and inflammatory conditions, fibroids the most frequent ailment. In America there are but few abdominal surgeons who have not a series of 100 or more cases of hysterectomy for myomata. In the years from 1894 to 1900 Kelly alone has operated upon 307 myomatous uteri. The prevailing fad is hysterectomy, and one may safely estimate that in the last four or five years American surgeons have operated upon from 50 per cent. to 75 per cent. of myomata discovered.

ETIOLOGY.—**Age**: The center about which the myoma develops probably exists in fetal life, yet the tumor does not attain pathological importance until late in the sexual life. It is very exceptional to meet with these tumors in the adolescent. They have been found in children, and Wells reports a case of fibrocystic uterus in which the history warrants the belief that the tumor was present when the girl was fifteen years of age. In 1,762 cases, reports of different writers, Roger Williams found only

26 instances of myomata in those under the age of twenty years. In 1898, Spencer was able to collect only 40 cases in women under twenty-five years of age. They are most frequently recognized clinically between the ages of thirty-five and forty-five years. The appearance of symptoms does not indicate the age of the neoplasm. Their development, reckoned from the appearance of symptoms, is not infrequently noticed after the fiftieth year and occasionally in women over sixty years.

Race: The negro women of the Southern States are frequent subjects of myomatous disease of the uterus. I can safely assert that fully 50 per cent. of all these women over forty years of age presenting at the gynecological clinic bear fibroids of the uterus. There are no observations reliably establishing the relative frequency in the white and black.

Heredity: By many heredity is advanced as bearing etiological claims; and instances, such as cited by Beyea, in which uterine fibroids appeared in four sisters, mother, grandmother and aunt, are striking but too rare to be conclusive.

By Roger Williams developmental irregularities are considered as the chief morphological factor in the development of myoma. Their occurrence where are found such deformities as uterus unicornis, uterus duplex, absence of vagina, and infantile, undeveloped uterus (Sutton), and their frequent association with dermoid, intraligamentary and other cysts, lend color to this position of Williams. Cohnheim's theory that tumors originate in rests or developmental defects seems, in a general way, established. Its application to the histogenesis of uterine myoma has received much consideration and high endorsement. First Babes, then Recklinghausen, and later Ricker found in these tumors the primitive epithelium of Müller's duct and abnormal ingrowths of muscle-cells around the rudiments of Wolffian ducts. Meyer showed glandular structure in the muscular tissue of the uterus in new-born children. Scattered epithelial elements may come from sequestrated processes of the glands of the mucosa extending into the myometrium, or from the remnant of the Müllerian duct.

Landau cautions that we must not mistake the adenomyoma, a tumor in which there is proliferation of epithelial elements as well as muscular, for the globular histoid myoma. To determine

the histogenesis of myoma and its relation to Wolffian or Müllerian rests Cohen examined the whole of many small tumors by means of serial sections. In none of the specimens were there epithelial inclusions found, and he claims that this demonstrates the fallacy of Ricker's hypothesis that all myomata of the uterus originate in adenomata.

In these investigations he found that the muscular fibers of the neoplasm were arranged concentrically around a small artery, an observation made by Kleinwächter in 1883, who said that in the tiniest myomata there were minute capillaries surrounded by round cells, and these, changing, finally resembled muscle-cells. This view of the arterial origin of myomata, either from arteries or arterioles, is upheld by Pilliet. And Gottschalk concludes that fibromyomata have their origin in long-continued irritation, and that their development is dependent directly upon arterial blood-supply. Santi asserts that he finds no fact to convince him that their origin is from the vascular tunics, and that the assertion of Rösger that the arrangement of the fibers about the vessels is proof of their origin from the muscular coats is erroneous, since there are more circular fibers than longitudinal in the vessel-walls, and a myoma arranges itself against the prevailing tendency. Furthermore, the absence of elastic fibers in myomata is one of the strongest facts against the origin from the vessel-wall.

A blanket-theory, expressed and apparently somewhat generally received at the Eighth German Gynecological Congress, 1889, is that uterine myomata take origin from the muscular coats of the small uterine arteries. These vessels pass directly into the zone of tissue so very rich in elastic fibers surrounding the neoplastic nodule, which grows by apposition of new strata of muscle arising from the peripheric vessels. This process is a pathologic expression of the formative activity innate in the general system, having its most evident manifestation in the uterus, a view held by Strassman and Lehman, who claim that the alterations in the heart, vessels, and uterus all proceed from a single affection causing vasomotor disturbance.

Another view which confines the formation of myoma to causes inherent in the uterus is thus expressed by Mann: It is the innate nature of the uterus to grow, stimulated by preg-

nancy; if that stimulus is not applied the atypical growth takes place. Routh, also, looks upon it as a sort of artificial gestation.

While congestion and the many conditions giving rise to it have long been recognized as elements in the development of myoma, there has, as yet, been no conclusive proof of the germ-origin of these neoplasms. Dr. Mary Dixon Jones asserts that for a fibroid to develop the tissues of the uterus must first be reduced to medullary elements by inflammation, and that it does not depend upon the year, age, or period of life, but it is when there is infection and consequent inflammation; that it is this disease of the uterus that produces fibroid growths. Semb regards fibroid tumors of the uterus as originating from irritation, chiefly in the endometrium. Claisse, in his Thèse de Paris, 1900, says that these new formations, essentially inflammatory, develop at the expense of the endo- and perithelial elements; they are associated with analogous lesions of the peritoneum, adnexa, and mucosa; they entail a marked leucocytosis, and are probably due to microbes, the poisonous elements gaining entrance through the mucosa.

Nothing definite is known concerning the origin of fibroid tumors of the uterus. It seems probable that in all theories there is an element of truth, and from the study of the subject the author concludes that myomata grow from embryonic foci in the walls of the uterus, which residua are frequently found in immediate relation with small arteries; and these congenital foci are awakened into activity by conditions disturbing the circulation, such as is supplied by inflammation, displacements, traumatisms, sepsis, and pregnancy.

Pathology.—In the primitive stage fibromyomata consist of an aggregation of round cells transforming themselves into spindle-cells and then assuming the form of smooth muscle-cells, larger than those of the surrounding muscular tissue of the uterus. They arrange themselves in a ring and form a whirl or kink in the fibers of the muscle. Usually they are arranged concentrically about small arterioles. These central vessels may at first dilate, but they soon become obliterated. The fibers interlace in all directions and in many degrees of density. In pure myomata no other kind of cells or fibers than muscular ones are seen, but with the growth and development of the tumor the groups

of muscle-cells are blended with or completely separated by conspicuous tracts of fibrous tissue.

The microscopical picture varies with the parts of the tumor chosen for section. The existence of a central vessel, so stoutly maintained by Gusserow and Kleinwächter as constantly present in the tiniest myoma, is denied by many careful observers. The myomatous nodule, which Roger Williams likens to a small knot in a piece of wood, lies in a bed of connective tissue which is proper to the uterus and which separates the nodule from the surrounding fasciculi. As the tumor enlarges, this tissue is compressed into a fibrous covering or capsule, from which the little nodules may be picked out or large tumors enucleated without disturbing the surrounding muscular structure of the uterus. Fibrous bands from the capsule dip down into the internodular spaces and may render difficult the freeing of the capsule. The blood-supply is not derived from the central vessel but from a wreath of anastomosing vessels, which penetrate its capsule and plunge into the center of the tumor. Veins, lymphatics, and nerves accompany the arteries in their course between the fibrous bundles. The capsule and periphery of the tumor receive a greater blood-supply than does its central portion. Beneath the capsule large arteries are sometimes found and occasionally penetrate deeply into the substance of the tumor, and in such cases the veins are disproportionately larger. This great vascularity is especially true in myoma complicating pregnancy.

When very small and of recent formation these tumors consist chiefly of muscle-cells, but it is quite exceptional for a tumor to attain to any considerable dimensions without a greater or less proportion of fibrous elements. If the muscular tissue predominates the growth is softer and more vascular, the capsule is not so well marked; indeed, a hyperplasia of the muscular tissue of the whole or a part of the uterus is said to occur, which is spoken of as a diffuse myoma; I have never seen such a specimen. Generally speaking, the fibrous elements increase with age, and hence old tumors, uncomplicated, are usually hard.

There is no condition, says Kleinwächter, in the growth of fibroma of the uterus sufficiently strongly marked and regular to enable one to determine the age of the tumor. This observation was made after a careful study of 40 selected cases. The stereo-

typed statement of text-books that fibromyomata grow very slowly and cease growing after the menopause is refuted by daily clinical experience. I removed by *morcellement* through the vagina from a woman fifty years of age a tumor weighing four pounds. After its enucleation I discovered in the opposite wall of the uterus a tumor the size of a hen's egg. Three years later this tumor was enucleated in a similar manner, having attained to the size of a cocoanut, and weighing two pounds. The fact is that the behavior of these tumors is contingent upon so many conditions, intrinsic and extrinsic, that there is no prescribed rule regulating their growth. Those composed chiefly of muscular elements are usually more vascular and grow more rapidly than the fibroid sort. Wylie seems to think that, like other organic structures, they have a period of growth, repose and decay. Appearing usually after the age of thirty years, they continue more or less actively to grow until the menopause, when they may subside or, perhaps quite as frequently, undergoing degenerative changes, increase very rapidly in size. The rapid enlargement is not necessarily due to growth of the constituent elements, but may be owing to changes in the blood-supply, as in pregnancy, or to edema, cystic degeneration or other changes. One peculiar characteristic occurring in the life-history is their tendency to grow to a certain size, then to remain stationary or even to regress somewhat, later to take on active development. This behavior is not always contingent upon the menopause. And while in certain instances retrogression after the menopause is observed, the old teaching that this should be the expected course is wofully misleading and dangerous. Fibroids have been known to disappear; Doran has collected 37 instances of the partial or entire disappearance of fibroids, 13 of these directly connected with pregnancy. In one case in which I attempted hysterectomy, concluding that it would be unsafe to remove the tumor, which was larger than a child's head, I abandoned the operation. Seven years later an examination showed the tumor to be densely hard and no larger than one's fist. In this case the menopause played its assigned part.

Their size varies from a minute nodule to a colossal tumor overdistending the abdomen. I have removed for Dr. R. F. Boyd, of Nashville, from a negress, aged forty-two years, a large

rapidly-growing myoma weighing eighty-two pounds; recovery was uninterrupted. Hunter exhibited to the Obstetrical Society of New York a specimen weighing one hundred and forty pounds. It is now quite exceptional to meet with a tumor weighing over twenty pounds—surgeons are too alert.

Soft myomatous tumors frequently occur singly. Those more fibroid in character are usually multiple. The clinical appearance of one tumor does not indicate its isolated existence; many seedlings may be scattered throughout the uterine walls, a fact that the advocates of myomectomy somewhat ignore. Sutton has lately described microscopical sections taken from a uterus which contained one hundred and twenty minute fibroids.

The common site is the body of the uterus. They grow more frequently from the posterior than from the anterior wall, and more frequently from the fundus than from the cervix. The relative frequency of cervical fibroids to those of other parts of the uterus is estimated at from 4 per cent. (Löhlein) to 12½ per cent. (Mundé). Hart and Barbour say that cervical fibroids do not occur oftener than 5 times in 100 cases, but that when fibroids complicate pregnancy location in the cervix is 20 out of 100. Cervical fibroids are of comparatively small size, yet Amann removed a tumor weighing twenty-five pounds which developed in the cervix entirely in front of the peritoneum. Especially dangerous and difficult of management are those fibroids that grow from the anterior wall under the attachment of the bladder, pushing that organ upward before them.

All myomata originate in the musculature of the uterus, and in that sense are primarily interstitial. From their seat and direction of development we recognize three varieties: (1) A tumor developing in the wall of the uterus and surrounded on all sides by muscular substance is designated an *interstitial, intramural,* or *intraparietal* tumor. (2) If, in its growth, meeting with less resistance, it advances toward the mucous surface and projects into the uterine cavity, a greater portion of its circumference being covered by endometrium, it is designated a *submucous* myoma. (3) More frequently the direction of growth is toward the abdominal cavity, the tumor lying just beneath the peritoneum, when it is designated a *subserous* fibroid.

1. Interstitial fibromyomata in structure more closely resemble the uterine tissue. Their growth is often rapid and symmetrical, and their vascularity greater. They may encroach equally upon the submucous and peritoneal surfaces. Interstitial cervical tumors, moulded by their surroundings, present a barrel-shape. They often form large, smooth, soft tumors, greatly lengthening the cavity of the uterus. If of small size, their number is usually multiple (*vide supra,* Sutton's case). When very vascular they are designated *angiomyomata* or *myomata telangiectodes.* It must not be inferred that all interstitial tumors are of this soft sort. Roberts says that they are usually of the hard, fibrous variety.

2. If the direction of the growth is toward the cavity of the uterus, the tumor may project into the cavity, the growth of the tumor and the contraction of the uterine muscles tending to extrude it into and from the uterus. These tumors are not supplied with a dense fibrous capsule, but are covered by mucous membrane which retains its normal characteristics until it descends into the vagina, when the ciliated epithelium is replaced by squamous cells. Submucous pedunculated fibroids are designated uterine polypi. Their favorite seat of attachment is the fundus. They may be soft when sessile and tend to assume an ovoid or pyriform shape, moulded by the contractions of the uterus. If slow of growth they are densely hard and fibrous. As a rule, submucous fibroids are of firm, almost tendinous consistency so long as they remain in the uterine cavity. When they are extruded into the vagina they undergo softening, become edematous, and suppuration and gangrene may occur. Cervical submucous tumors, unless complicated by pregnancy, are of little importance, as they often grow through the os uteri and by their pedicles hang into the vagina; or, if sessile, they are easy of enucleation.

3. Subperitoneal fibroids are frequently multiple, may be sessile or pedunculated. When stalked, Virchow designated them peritoneal polypi, but the term 'polypus' should be reserved for tumors that grow into the cavity of the uterus. As these tumors by their direcion of development restrict their vascular supply, they are usually hard and fibrous in character, of slower growth

than the intramural variety, and are especially liable to certain complications.

All varieties of fibroids may be met with in a single uterus. Uterine polypi more frequently exist singly than do the other varieties. The pathological and clinical importance of fibroids depends as much upon their location as upon their size and rapidity of growths. Fibroids separate the folds of the broad ligaments. These intraligamentous tumors, by their position and impaction in the pelvis, are of greatest clinical importance. A small subserous tumor, pedunculated, may become imprisoned in the pelvis under the promontory of the sacrum and excite urgent pressure-effects. Occasionally subserous tumors so imprisoned force the uterus into procidentia. It is of the greatest importance to note the position of the uterus with relation to the neoplasm. Polypoid growths drag the entire uterus into descensus and not infrequently fundal polypi produce inversion, a fact that must be borne in mind in removing them. Intraligamentary fibroids and those growing in the lower portion of the body of the uterus and lying in the pelvis crowd that organ to the opposite side, and frequently elevate it out of the pelvis. A mural fibroid of the cervix distorts the vaginal cervix and os externum so that that opening appears not unlike a quarter-moon. Submucous, sessile or pedunculated, and single interstitial myomata symmetrically enlarge the uterus, forming a central or hypogastric tumor. Long pedunculated subserous tumors usually draw the uterus upward, elongating it and distorting its cavity.

Rotation of the tumor upon its pedicle may occur; such an accident may result in gangrene of the tumor. My first hysterectomy was for this condition. From slow torsion the pedicle may atrophy and separate from the uterus entirely. Usually, however, inflammatory attachments are acquired, chiefly to the omentum, from which organ sufficient blood-supply is obtained for nourishment. These sequestrated or parasitic myomata are often calcified. Torsion of the uterus as a pedicle from rotation of subserous fibroids has been observed.

Multinodular tumors may efface all the outlines of the uterus. Some may lie intraperitoneally, others between the folds of the broad ligament, and still others, developing downward, encroach upon the rectovaginal septum and occlude the vaginal canal.

Diffuse rapidly-growing interstitial myoma shows a tendency to assume the shape of the gravid uterus, even up to full term. While fibromyomata are generally globular in shape, they are moulded largely by their surroundings. Kelly illustrates one that conforms very perfectly to the curves of the sacrum, showing the promontory.

A general hypertrophy of the uterine wall attends the growth of myoma. Old tumors, especially of the subserous variety, not infrequently produce atrophy of the uterus, the circulation of the organ being interfered with by traction or pressure. In submucous tumors the opposing wall is usually thin. The cavity of the uterus is enlarged but occluded by submucous tumors. Multiple tumors, interstitial or subperitoneal, distort the uterine cavity and it is difficult or impossible to pass the sound. In single mural growths the depth of the uterine cavity is sometimes enormously increased.

To those who see in inflammation the essential cause of myoma there is always associated with the neoplastic growth metritis and endometritis. In many cases of myomatous uteri there is absolutely no endometritis. The mucosa, like other surrounding tissues, may undergo hypertrophy, both glandular and interstitial. This overgrowth of its elements is not necessarily inflammatory in character. The presence of the tumor and the mechanical changes in circulation incident to its pressure are certainly great predisposing causes to infection. More frequently the condition of the endometrium is one of atrophy over the tumor surrounded by a corona of hypertrophy. The influence of the tumor, says Bishop, upon the mucosa depends entirely upon its position in the uterine wall and upon its size. If the tumor has reached a certain size it induces through mechanical influences a stretching and atrophy of the mucosa. The nearer the tumor is to the mucosa the greater the changes in that structure.

In 11 cases of Borisoff, in which there was atrophy of the endometrium, hemorrhage was a conspicuous symptom. In the mural tumors the vessels beneath the endometrium may be dilated, and extravasations of blood into the muscular substance sometimes occur. If the growth advances toward the mucosa, then sclerosis of the overlying mucosa follows. The hemorrhage is not from the tumor but from the degenerated and dilated veins sur-

rounding the dome of atrophied area. The submucous tumor suffers from erosion and necrosis of its endometrium. In subserous tumors the endometrium may show no alteration whatsoever.

In fibroma uteri the tubes and ovaries are always altered. The changes in the tubes simulate those that occur in the uterus. There is always hyperplasia of the muscular fibers and of the mucosa, chiefly of the latter. The changes in the ovaries are microscopical, consisting of proliferation of connective tissue and degeneration of blood-vessels with diminution of primordial follicles. By pressure the ovaries are frequently atrophied (Greio).

Complicating Pathology.— **Changes in the Tumor:** (a) *Edema:* One of the most frequent changes met with in fibroids, especially those that are pedicled, is edema. It is a nutritive change consequent upon some interference with the circulation. The tumor loses its firm consistency, and the tissues are everywhere in a state of liquefaction. Spaces exist throughout the tumor filled with yellow serum, and the tissues in contact with the fluid present a hyaline appearance. Doran believes that the edematous fibroid seen in women who have reached the menopause is a very different growth from one that has become edematous from compression.

(b) *Myxomatous* and other *Cystic Changes:* Myxomatous degeneration is possibly an advanced stage of edema. Large tumors are especially predisposed to this change. In their center are areas of tissue that have been converted into mucin. Large rod-like bodies with rounded extremities, similar to the nuclei of non-striated muscle-cells, are found amidst the hyaline structure (Rutherford). The collection of fluid in these tumors gives us one form of fibrocystic tumor. Homans has created some confusion in stating that by fibrocystic tumor he means one whose outer surface is covered with cysts having rather thin walls, and not those tumors that have smooth outer surface whose interior contains dilated lymphatics. It has been my privilege to deal with many fibroid tumors surgically. I have frequently found little serous cysts which I attributed to peritoneal inflammation, but I have never encountered Homans' true surface fibrocysts. A fibroid may become cystic through myxomatous degeneration. Only occasionally do we find cysts with endothelial lining, due

to dilatation of lymphatics; such tumors are designated lymphangiectatic myomata. Certain soft myomata, very vascular, with cavernous spaces filled with blood, are called angio- or cavernous myomata. When a cyst contains large quantities of blood the question naturally arises as to whether it is a telangiectatic fibroid, a sarcomatous degeneration, or whether it is retained menstrual blood, the cavity of the uterus communicating with the fibroid through the Fallopian tube. The cystic changes ordinarily encountered in fibroids are either myxomatous, lymphangiectatic, or telangiectatic. Recent work has attempted to show that the genesis of fibrocysts is from embryonic epithelial inclusion. Schauta says that occasionally a rare form of subperitoneal cysts, in which the cavity is lined with ciliated epithelium, is encountered, and he further calls attention to the fact that these cysts are always of the subserous variety, the form referred to by Homans.

A tumor undergoing cystic changes usually increases rapidly in size, is liable to the accidents of edema, infection, with suppuration or gangrene. This complication confuses diagnosis, since they are often mistaken for ovarian cysts. I removed a large subperitoneal fibroid some years ago that contained two gallons of purulent matter; the walls were a half-inch thick, the appendages apparently normal.

(c) *Fatty Degeneration:* Roberts refers to one specimen of fatty degeneration of a fibroid in St. Bartholomew's Hospital Museum. In 600 specimens Cullen found only one that had undergone fatty change. Brünings reports the removal of a tumor about the size of a child's head which showed fatty degeneration. This change is very infrequent.

(d) *Calcification:* This is perhaps the most common change that fibroids exhibit. It occurs in circumscribed areas, usually in the center of the tumor; and naturally it is to be found in aged tumors. The deposit takes place in a regular manner corresponding to the disposition of its fibers. In a tumor undergoing calcification, if macerated and freed from its organic matter, a skeleton consisting of calcium carbonate and phosphate will remain. Pedunculated subserous tumors are especially prone to this change, and by torsion of the pedicle the stony tumor may become detached. Cullen removed from the cavity of the uterus

a half-shell of calcified myoma, and refers to a similar case reported by Thorn. Calcareous changes are not uncommon in the central portion of the tumor. Should they proceed to any extent the tumor decreases in size and often remains inoffensive, yet Richardson reports a case of calcified myoma obliterating the ureter. Edebohls has recently removed 3 calcified fibroids by enucleation.

(e) *Necrobiosis* is a nutritive change brought about by gradual obliteration of the blood-supply. The tissues become soft, of a reddish-brown color, and are especially liable to infection and other changes.

(f) *Suppuration* and *Gangrene:* In 218 patients Noble met with necrosis in 12 instances. Inflammation, suppuration, and gangrene are common occurrences in submucous tumors. In interstitial and subperitoneal tumors they are rarer accidents. Torsion of the pedicle, traumatisms, injuries with infection in labor, and curettage are the common causes. The infection of the fibroid tumor may occur through the endometrium, or from adherent bowel. It pursues an especially malignant course if its occurrence dates to abortion or labor. Many inflamed fibroids are mistaken for sarcomatous tumors. When infection occurs it may only involve the capsule in part or as a whole, or central suppuration may occur. Freund refers especially to those cases in which necrosis is slow, the patient giving evidence of mild auto-intoxication. In such cases the necrotic foci are generally in the substance of the tumor. In the area infected pyogenic bacteria are found, usually the colon-bacillus. When infection occurs there is generally rapid increase in the size of the tumor, and if the process is virulent the symptoms of peritoneal involvement and systemic poisoning rapidly develop. In more chronic cases the inflamed tumor may acquire attachments, and through sloughing be discharged through the abdominal wall or into the bowel, as in Hall's case.

(g) *Torsion of the pedicle* in subperitoneal fibroids may occur, resulting in partial obstruction to the circulation with edema, inflammation, and the acquirement of adventitious adhesions, through which adhesions the tumor may be saved from death with its constricted uterine attachment atrophied or entirely severed. Should the axial rotation cut off the arterial supply,

gangrene may result. I removed a tumor weighing ten pounds that was apparently in a state of necrosis from complete constriction of the pedicle by several turns; there were no adhesions; a serofibrinous fluid filled the peritoneal cavity, and there was abundant deposit of lymph over the intestines; complete hysterectomy, thorough irrigation, and vaginal drainage saved the patient.

(h) *Malignant Changes:* There are two ancient ideas that are still extant: the first is that fibroids and carcinomata are antagonistic and do not often occur in association; the second is that carcinoma is not an accidental association with fibroma, but the result of a glandular endometritis due to the irritation of the fibroid. The association is not a common one; Mackenrodt met with only 8 cases of carcinomata in 104 cases of myomata. In 159 cases of cancer of the uterus there were only 12.5 per cent. of fibroids; and in four-fifths of the cases the neoplasms were entirely independent of each other (Williams). When cancer occurs it is either by the growth of epithelium from utricular glands into the fibroid or by cancerous proliferation of the epithelial inclusions sometimes found in myomata. It is scarcely probable, certainly not proved, that metaplasia of myocytes into cancer-cells occurs. The occurrence of sarcoma as a degenerative process, a sequel to fibroma, seems to be established by the records of many carefully studied cases; but here, too, the genetic relation is not established, and Sutton regards the question as somewhat in a "cloud."

The most important complications arising in the course of fibroids are those attributable to pressure. These tumors displace viscera and inhibit their function by compression; in this way the bladder and rectum are chiefly interfered with. They rarely give rise to intestinal obstruction, but when of large size they interfere with all of the abdominal viscera and the function of the diaphragm. The ureter is especially endangered by their presence. In incarcerated pelvic fibroids and in the intraligamentary sort this duct may be compressed, or so enclosed in the growth and constricted by it that permanent occlusion follows. In larger growths the pressure upon the ureter is exerted especially at the pelvic brim. Hydro-ureter and cystic kidney, pyo- or hydronephrosis, are the results of the ureteral obstruction.

The peritoneum is exceedingly tolerant of the presence of fibroma. Hydroperitoneum is rarely observed in the usual cases unless the tumor itself is undergoing some change.

Adhesions are likewise infrequent in uncomplicated fibroids, but when these growths are associated with tubal and ovarian inflammation the adhesions constitute a most important part of the pathology, add greatly to the suffering of the patient, and render difficult and dangerous any operative procedure.

Tubal disease, as we have shown, in its milder forms is present in about 50 per cent. of the cases of uterine fibromata. Hydrosalpinx is especially common. Pyosalpinx and ovarian abscess, though comparatively infrequent, are not unexpected complications. The presence of these inflammatory conditions of the appendages, with their associate peritoneal lesions, can usually be recognized clinically, and the distress they entail, to say nothing of the immediate dangers to life, divests fibroids of their innocence and places them among the most serious ailments afflicting women. The frequent association of fibromyomata with cysts, ovarian and broad-ligament, is too common to be a mere concidence. They both have kindred origin, probably in developmental defects. The author has met with ovarian cysts, intraligamentary, and dermoid cysts complicating fibroids. Noble has shown how frequently these cystic growths have complicated his operations for fibroids. Boxall found a fibroid of the uterus, cyst of the ovary, and carcinoma of the Fallopian tube, all in one patient. The occurrence of ectopic gestation with myomatous disease of the uterus is sufficiently common to give to the neoplasm a causal position. The appendix may become infected through the fibroid that is undergoing inflammatory changes, or an inflamed appendix may engraft its infection upon a healthy fibroid.

Myomatous disease of the uterus does not rest with the complications limited to the pelvis and abdomen. Varicosities of the veins of the extremities and pelvis become one of the dangers due to the presence of myoma. Freund especially calls attention to the liability of these patients to suffer from secondary hemorrhage or embolism if subjected to operation. Cardiac complications are held in greater apprehension by English surgeons than by us. By direct pressure upon the diaphragm the thor-

acic viscera may be displaced, and cardiac and pulmonary embarrassment may follow. With large fibroid tumors, as in pregnancy, owing to the greater resistance, cardiac hypertrophy with subsequent dilatation and degeneration may become an absolute contraindication to interference. Hemorrhage and the muscular weakness consequent upon anemia may cause cardiac dilatation even in small tumors or polypi. No myomatous uterus should be dealt with surgically without the investigation of the circulatory apparatus. Though not demonstrable, there occur with fibroid tumors pathological conditions of the nervous system largely due to nutritive disorders, but in some measure reflex in character, which are of symptomatic importance.

Varied as their pathology is, there yet remains a complication sufficiently grave and frequent to justify its separate consideration. I refer to the coexistence of pregnancy and fibroids.

Pregnancy: The unproductiveness of married women with myomatous uteri is practically a universal observation. A striking exception are the views of Hofmeier, who holds that the great majority of fibroids have nothing to do with the prevention of conception, and who even claims that the presence of myoma in women who marry late in life rather favors conception. Williams estimates that only 2.5 per cent. of children are born to myomatous women, whereas in a general community the average per cent. is 4.6. In Kelly's 266 married women with myomatous uteri there was an average of 2.03 per cent. of pregnancies. Fränkel states that more than one third of patients having these tumors have during married life but one child, whereas in a series of 2,000 cases of women who had other pelvic diseases but 5 per cent. showed the same comparative sterility. Kattmann has investigated 400 myomata, and he says that they are more frequent in married women who have not borne children than in any other class of patients. Opinions differ as to the relation of myoma to sterility; those holding the inflammatory origin of fibroma maintain that the sterility produces the fibroma, while others regard the sterility an effect. Pinard says that out of 13,915 parturient women at the Baudelocque clinic only 84 had fibroma. These 84 cases were all primiparæ over thirty years of age or secundiparæ with first labor ten or twelve years previously. He holds prolonged uterine inactivity to be the real cause of fibroma.

It seems very fair to conclude that fibroids do not necessarily act as a bar to conception, but certainly myomatous women are not fruitful. Submucous fibroids have greatest influence in preventing conception; next in importance are the interstitial, and least effective are the subperitoneal. The clinical existence of fibroids may date from the occurrence of pregnancy.

Unfortunately interstitial tumors lying low down in the body of the uterus, either posteriorly or between the folds of the broad ligament, do not offer the hindrance to conception that one would suppose. When pregnancy occurs in cases of submucous fibroids the tendency is for gestation to be interrupted by hemorrhage. In interstitial and subperitoneal growths the effect of gestation is to increase the vascularity of the tumor and greatly to stimulate its growth. The dangers occurring from the presence of fibroids in pregnancy depend largely upon the site of the growth. Tumors of the anterior wall produce retrodisplacement of the uterus, and while the tumor may lie free, the pregnant uterus becomes incarcerated in the pelvis. Intrapelvic tumors, interstitial or subperitoneal, by their increased growth incarcerate the uterus, compress the urethra, bladder, and rectum, and obstruct the ureters; acting thus offensively, they create a marked tendency to abortion during the first twelve weeks of gestation. The progressive enlargement of both uterus and tumor must result in the escape of one or the other from the pelvis, should abortion not occur. If the tumor is released, pregnancy may continue uninterruptedly to a favorable termination, but unfortunately in the intrapelvic tumors, especially of the cervical and intraligamentous sort, the tumor underlies the uterus, and that gravid organ with the bladder attached is displaced upward and laterally out of the pelvis, so that even the os uteri cannot be felt. In interstitial tumors of the fundus and body the growth is very rapid, the intra-abdominal pressure is greatly increased, the nervous and digestive disturbances incident to pregnancy are greatly exaggerated. Dyspnea, due to pressure and cardiac inefficiency, becomes pronounced. Varicosities of the veins of the extremities and pelvis, jaundice and albuminuria, and withal a constant tendency to premature labor are the dangers during pregnancy. Subperitoneal tumors, when small, by their intrapelvic position, give trouble chiefly through their pressure-effects;

they do not become so edematous nor enlarge so rapidly under the influence of gestation as the other forms, yet they are liable to changes of edema, cystic degeneration, and to the accident of torsion of the pedicle or elongation or rotation of the uterus.

When abortion occurs as the result of the complication of fibroids with pregnancy the maternal mortality is 12 per cent. (Stavely). If the abortion is induced the mortality is more than double this per cent., about 33 per cent. Abortion is only likely to occur in submucous fibroids, and in those tumors that produce incarceration. The likelihood of abortion is not so great as is generally taught. In 20 cases under Hofmeier's observation there were only 2 premature deliveries. Should pregnancy advance to full term, Stavely found that in 597 cases in which nothing was done until labor came on there were 220 mothers lost. In delivery by the natural passage at term the maternal mortality varies from 25 per cent. to 55 per cent., and the fetal mortality is 77 per cent. (Lafour). Winckel states that pelvic presentations occurred in 23.5 per cent. in this complication. During labor the dangers are abnormal presentations, prolapse of cord and extremities, placenta previa, inefficient pains, hemorrhage, rupture of the uterus, and absolute occlusion of the birth-canal, rendering delivery impossible. In the third stage of labor hemorrhage chiefly threatens. After delivery involution is retarded and infection with softening of the tumor, general sepsis and early death, or else sloughing of the tumor with chronic sepsis and death from exhaustion are the dangers to be feared. Should the patient escape all these dangers, there not infrequently occurs after labor marked regression of the tumor, so that a growth that may be of alarming size during pregnancy may become, post partum, utterly insignificant.

There are several determinate points that present themselves in this subject of pregnancy complicating fibroids, the first of which is the recognition of the coexistence of the two conditions. The difficulties here rest largely upon the site of the tumor. In subperitoneal tumors the diagnosis is usually easily made; and so likewise in intraligamentous tumors. But in mural growths and those of cervical site the difficulties are greater. In women who are approaching the menopause and who are known to have long been possessed of a fibroid, the unexpected often happens.

Changes are observed in the tumor; its rapid growth is evident, softening occurs, and we are prone to ascribe these phenomena to degenerative processes. It is in these women, especially if they are newly married, that we not infrequently meet with pregnancy. We should not be content with simply recognizing the complication, but we should seek to determine the character of the growth, its relation to the uterus, the possibility of elevating it out of the pelvis, our ability by posture or mechanical means to relieve pressure-symptoms. We must, furthermore, watch its evolution to see that the uterus retains its axial relation to the pelvis, and while giving attention to the local behavior of the parts involved, the general condition of the patient must receive consideration. It is quite conceivable that, for reasons based upon systemic conditions, interference may be demanded.

Entertaining the most conservative views of the management of fibroids in pregnancy, holding that only under exceptional conditions do they demand interference, I none-the-less strongly advocate surgical relief at that time when most urgently demanded and before the patient has reached the state through suffering, exhaustion, and auto-intoxication when the most skilful procedure must prove unavailing. The general indication for government in these cases is a humane regard for both lives, but that of the mother must ever take precedence. An induced abortion sacrifices the fetus and exposes the mother to dangers even greater than pan-hysterectomy, thwarts the influence of normal involution upon the tumor, and is wholly unjustifiable. Polypoid growths, though infrequently associated with pregnancy, on account of the hemorrhage and painful contractions they induce should be promptly removed. Cervical tumors offer the greatest obstruction to labor; they enlarge rapidly, and their pressure-symptoms demand relief. If it is obvious that the tumor will prove an obstruction to delivery, vaginal enucleation should be done, when possible, prior to the fourth month. If this does not seem feasible we should consider the possibilities of permitting the case to advance until the child is safely viable, when Cæsarean section followed by complete hysterectomy is the preferable procedure. Those cervical and body-tumors that grow between the folds of the broad ligament may, by their rapid enlargement, give rise to such pressure-symptoms that we are not war-

ranted, in the interest of the mother, in delaying operation. Hysterectomy should be done in all those cases in which independently of pregnancy relief is urgently demanded. This will embrace all conditions dangerously affecting the tumor itself, the adnexa, the contents of the uterus, such as the death of the fetus, and those local conditions attended by unbearable suffering incident to pressure; and furthermore, much to be emphasized, the occurrence of lesions in remote organs and systemic conditions imperiling life. The mere growth of the tumor is not in itself a warrant for intervention. The pelvic position of the tumor should not lead us to decide hastily upon operation; the resources of nature are great, and the sudden release of a seemingly hopelessly incarcerated tumor or uterus may occur. The dangers anticipated in labor should bide their coming. When it is apparent that delivery is impossible, we should elect a suitable time, after the child is viable, for Cæsarean section, to be followed by hysterectomy. To this there is a luminous exception: when fibroids, pregnancy, and placenta previa are found, complete hysterectomy is immediately and imperatively demanded. A knowledge of and speculation upon post parturient complications should not be grounds for interference. After delivery we may be called upon to operate for uncontrollable hemorrhage, for sloughing of the tumor, or for infection. Myomectomy during pregnancy is a serious procedure, and if my experience were sufficient to justify, I would say a questionable procedure—this, too, in the face of many happy results in this country and a knowledge of Martin's statistics. When so-called conservatism reaches that extreme degree which induced a Philadelphia surgeon to remove 11 fibroids by myomectomy, and an ovarian cyst at the same time, leaving a pregnant uterus, the patient aborting in eighteen hours, we heartily endorse the severe rebuke administered by Montgomery. Rosenberg considers myomectomy utterly unjustifiable during pregnancy; this position can scarcely be maintained in the face of the returns. And when so high an authority as Martin, the originator of the method, says that in case of dystocia from fibroids Cæsarean section, followed by myomectomy, is the operation of choice, total or partial abdominal hysterectomy being undertaken only as an operation of necessity, one is not warranted in excluding it from the list of justifiable procedures. Yet

fear of that interminable oozing, which we sometimes encounter in myomectomy upon the non-pregnant uterus, would lead me to choose very carefully my cases for myomectomy. Total hysterectomy for myomata complicating pregnancy in Landau's clinic since 1885 has given a mortality of 8.9 per cent.; supravaginal amputation, 11 per cent. In this country supravaginal amputation is the more popular procedure.

SYMPTOMS.—The symptoms presented in the life-history of fibromyomata of the uterus are attributable to the presence of the tumor itself, to the changes occurring in the tumor, and to the extraneous complicating pathological conditions. Varied as these several factors are, it manifestly follows that such conditions will in divers ways express the complaint of the patient. There are no symptoms pathognostic of myoma, no recital of a group of symptoms clearly indicative of its presence, yet when one has studied a large series of cases he finds that, though in individual cases the behavior may be exceedingly erratic and peculiar, their general deportment evinces the family characteristics.

Pain: It can scarcely be said that pain is characteristic of fibroids, but if modified and expressed as a feeling of discomfort, a sense of weight, bearing-down, and heaviness, we find it the most constant of the early symptoms. Real pain, acute suffering, is a symptom of some complication or peculiar behavior of the tumor. It therefore follows that in the history of a fibroid tumor the quality of pain may change with the development of the growth. The pain of pressure is the only suffering that an uncomplicated fibroma will occasion; the part compressed does not yield the same character of pain. In intrapelvic tumors the pressure upon the sacral nerves may cause a constant ache. If the tumor compresses the bladder pain may only be felt when that organ is distended or in the act of emptying itself. And the same may be said of the rectum. Those tumors situated on the anterior wall of the lower segment of the uterus displace the bladder upward, compress the ureter, occasion frequent micturition and great dysuria. In intraligamentous tumors that force the uterus out of the pelvis, vesical and rectal pain are often the most distressing symptoms. Large tumors that rest upon the brim of the pelvis have more accommodating surroundings, and

the discomfort they occasion is the embarrassment of function due to their actual size and weight. The pain of pressure is constant, increased by exercise, relieved by rest and posture; it augments with the approach of menstruation, and is worse when constipation exists. It is relieved by hemorrhage and by purgation. The pain of pressure may be referred along the course of the nerves to the genitals and thighs. The most intractable sciatica is sometimes occasioned by intrapelvic fibroids. The greatest pain may be in the iliac region from compression of the ovary. Rectal tenesmus and pain in the absence of any local diseases in the bowel should suggest the possible presence of a fibroid. Pressure may occasion referred pain in an entirely different way. Those tumors, the intraligamentous especially, which involve the ureters, may occasion but slight pelvic pain; but through such obstruction and the consequent hydronephrosis the pain is felt in the sacral region. Abdominal tumors, by the displacement of viscera, cause difficult digestion with local or general abdominal pain. Submucous fibroids excite resentment on the part of the sensitive endometrium and stimulate uterine contractions. Such tumors, however small, cause expulsive labor-like pains; when of large size and engaged in the os there is the unabating pain of pressure. An incarcerated pelvic tumor becomes edematous, impedes the circulation of all surrounding viscera, occasions congestion, invites infection, and always causes acute suffering. Paroxysmal pain sometimes occurs in other than in polypoid tumors. Subserous pedunculated tumors often rotate about their pedicles, or, as we have shown, may occasionally twist the uterus upon itself. The obstruction to the circulation usually excites severe paroxysms of pain, felt in the abdomen, and not attended with the expulsive bearing-down effort so characteristic of polypi.

When pain develops in a previously inoffensive fibroid it is usually indicative of some change in the tumor itself or its surroundings. Cystic degeneration, though a comparatively slow process, always gives rise to greater discomfort. Infectious processes involving the tumor itself, uterus, or appendages, are always attended by severe pain; and as the peritoneum participates in the inflammation, the suffering is acute. The traction upon adhesions excites pain. Those patients who have peritoneal ad-

hesions are usually debarred from exercise of any kind. Sexual intercourse may be painful because of obstruction or hypersensitiveness. Should pregnancy occur the rapid growth of the tumor and uterus adds greatly to the patient's discomfort. The frequent occurrence of paroxysmal pains denotes the tendency to abortion. Should pregnancy advance to full term, our text-books tell us that labor pains are weak and inefficient. This may be the rule, but in one case, at least, I am informed by the physicians in attendance that for seventy-two hours the pains were rhythmical and terrific, wholly ineffectual because of the complete blocking of the birth-canal by the tumor.

In reciting those conditions in which pain is a prominent symptom of fibromyoma of the uterus, it is not intended to emphasize too strongly pain as a constant symptom. Many fibroids are borne with grace and comfort.

Menstrual Disorders: The most constant menstrual disorder resulting from fibroid tumors of the uterus in those who have reached the proper age is the delay of the menopause from two to five years beyond the usual time.

A free menstrual discharge, lasting longer than the usual time, may appear as a symptom of myoma before the tumor has attained to appreciable size. The occurrence of uterine hemorrhage is therefore regarded as one of the initial symptoms of this disease. Fully 80 per cent. of myomatous uteri show menstrual disorders. The bleeding at first occurs at the regular catamenial time. It may continue to observe this regularity, but intercurrent hemorrhages become a feature of certain forms of tumor. The nearer the tumor lies in relation to the mucosa the more profuse and irregular the bleeding. It therefore follows that mural and submucous tumors, the latter especially, are attended by hemorrhage. Subperitoneal tumors, especially of the multinodular type, may not only be unattended by excessive menstruation, but in some instances they actually produce amenorrhea. When hemorrhage occurs in multiple subperitoneal tumors, it is not infrequently due to a small undiscovered submucous growth.

To appreciate fully the causes of hemorrhage we must recall the changes that take place in the endometrium. In all growths which enlarge the cavity of the uterus there is a greater expanse

of surface influenced by the turgescence of menstruation. When the normal anastomosis of the uterine vessels is deranged by the growth of a myoma, the efferent veins do not promptly carry the arterial blood away from the uterus. This venous stasis becomes more marked, of course, at each menstrual period, and it is expressed by a freer and more prolonged hemorrhagic discharge. Unless the mechanical obstruction offered by the tumor is very great, collateral vessels dispose of the excessive accumulation and menstruation continues normally; so that in many myomata, interstitial and subperitoneal, there is not necessarily a menorrhagia. But, as Clark says, "with the encroachment of the tumor upon the uterine cavity, a second and most weighty cause of hemorrhage comes into action. . . . A thinning of the mucosa and a coincident degeneration of the vessels renders the usual diapedesis much easier or gives rise to an escape of blood through actual rupture of the capillaries. . . . In further expulsion of the tumor the vessels undergo actual necrosis along with the tumor, which renders them brittle and more liable to hemorrhage through extensive rupture." I have thus quoted freely from Clark as to the causes of hemorrhage since his study of 100 cases as to the sources of bleeding in myomatous disease of the uterus represents the most original and thorough work with which I am acquainted. The bleeding in myoma uteri rarely comes from the tumor or its capsule, but from the engorged vessels of the endometrial zone encircling the dome of the tumor or its pedicle. Should necrosis of the tumor occur, large sinuses may be opened by sloughing, and sudden, alarming hemorrhage result. Intercurrent hemorrhages, metrorrhagia, indicate a greater degeneration of the mucosa and are usually present in polypi and in large submucous tumors. Menorrhagia and metrorrhagia are, therefore, symptoms of fibroids that are advancing in their growth upon the endometrium. In pediculate subperitoneal tumors the lining of the womb's cavity may be normal, and such tumors rarely give rise to hemorrhage.

The bleeding in myomatous disease is sometimes copious, often alarming to the patient, yet rarely fatal. When it appears as a simple menorrhagia many patients experience great relief from their feeling of weight and heaviness, but the ultimate result from this periodic copious loss of blood or the constant intermenstrual

trickling of metrorrhagia is to bring about a condition of anemia. The patients grow pale, have a waxy look, are not infrequently fat and flabby; their general muscular system shows relaxation and degeneration; the heart, especially, suffers from fatty degeneration and dilatation.

Dysmenorrhea: When a woman suffers pain in connection with menstruation she may be said to be the subject of dysmenorrhea if that pain occurs within the genital sphere, which is that region bounded above by the iliac crest and below by the knee. But few women with fibroids can be said to have a normal menstruation. The pain excited by a fibroid during menstruation is somewhat difficult of explanation. It is not always obstructive, nor is it necessarily inflammatory in character. It is in a great measure, no doubt, spasmodic; the engorgement consequent upon approaching menstruation excites the uterus to labor. In submucous tumors the irritability is greater. It therefore follows that menstrual pain, like hemorrhage, is more conspicuous when the endometrium is involved. For several days prior to the appearance of menstruation a woman with a myomatous uterus has an exaggeration of all those symptoms which we have ascribed to pressure. These are relieved in a great measure by a free flow, but not entirely. This partial abatement of pain is significant, as it is one of the distinguishing points from other forms of dysmenorrhea. In multinodular fibroids, the sort that Price claims is so usually associated with disease of the appendages, there is frequently severe dysmenorrhea and not menorrhagia. The pain in these cases is attributable more to the diseased appendages than to the tumor.

Leucorrhea: Occasionally patients with fibroids suffer from a thin brown, watery discharge, hydrorrhea. It is common in those cases of large interstitial myomata which greatly increase the depth of the uterine cavity and cause a glandular endometritis. I have also seen it in old women with polypi. When endometritis is present as a complication, a mucopurulent leucorrhea is found. Foul discharges are usually due to the disintegration of submucous tumors; they may not infrequently excite suspicion of cancer or retained placenta.

Sterility: We have under the complication of pregnancy discussed the relation of sterility to fibroids, and reiteration of these views is not necessary here.

A woman, the subject of myomatous disease of the uterus, may suffer comparatively little pain. Menstrual disorders may be inconspicuous. Her linen may be unsoiled by vaginal discharges. She may be a virgin or a housewife with a multitude of children. By emphasizing the above individual symptoms it must not be inferred that they constitute the category of these patients' complaints. When the tumor assumes recognizable proportions the majority of the women are practically invalided. They suffer from a variety of reflex nervous phenomena, nausea, faulty digestion, flatulency, headache, dizziness, tachycardia arhythmia; not infrequently they are hysterical and visited with nervous storms; they are possessed of irritable tempers; they are incapable of exertion, and, weakened by anemia, they lead a useless and miserable life. Conscious of their burden and apprehensive of its results, they become melancholy and despondent. Should infection occur, septic symptoms are added to the general phenomena. Cystic changes produce rapid growth of the tumor and greatly impair the general health. The busy practical surgeon finds but little time to go into the minutiæ of the subjective symptoms. Much may be learned from their study that will have a very definite bearing upon the treatment. The diagnosis of fibroid tumors rests chiefly upon the physical signs, but the surgical treatment must find its warrant in a great measure in the subjective symptoms.

Physical Signs.—**Inspection:** When a myomatous uterus attains sufficient size for the tumor to rise above the brim of the pelvis, the morphology of the abdomen will depend upon the size, position, and number of the tumors, upon the thinness or obesity of the patient, and upon the association of ascites. Single tumors, intramural or submucous, produce a symmetrical swelling, first noticed in the hypogastric region. Large subperitoneal fibroids may occupy the central region. But usually the abdomen is asymmetrical in outline, and its general contour broken by the eminences of the multiple tumors. They may rise very abruptly from the pubes, and usually slope as acutely to the ensiform. They not infrequently have a distinctly lateral position. Intraligamentous tumors especially broaden into the iliac region. In thin nulliparous subjects the parietes may be tightly stretched over the tumor. If ascites, an infrequent complica-

tion, is present, the outlines of the tumor are obscured by the general abdominal fulness. The abdominal veins are not often enlarged. Of more significance is it to find the veins of the extremities varicose, with edema of the limbs, frequently unilateral. If hemorrhage is a conspicuous feature the skin has a pale, waxy look. There is not necessarily emaciation; indeed, many of these patients are fat. The neck, arms, and face are not thin as in ovarian tumors. With negroes especially we notice that the jaw is broad, cheek bones high, face square. I have seen this sufficiently often to admit that there is something characteristic in the "fibroid countenance." Fibroid polypi may protrude from the vaginal orifice, and if their attachment is fundal, carry with them the inverted uterus; or if held in the grasp of the cervix, on inspection we find edema of the labia, protruding hemorrhoids, ectropion of the urethral mucosa; and occasionally supravaginal cervical tumors force their way downward, separating the vagina and rectum or vagina and bladder, and cause bulging of the perineum. Presenting polypi are usually white and densely hard; the more vascular forms have generally undergone edema and necrosis, and appear as sloughing, gangrenous masses.

Palpation: Under palpation fibroids offer firm, unyielding, even hard resistance. Single tumors are smooth, globular in outline, and often resemble the pregnant uterus. Such uniformity, however, is exceptional. Irregular nodular masses may be felt upon some portion of the periphery of most single tumors. In multinodular fibroids of the subperitoneal type we may feel a conglomerate mass composed of a number of tumors of various sizes, forming bosses or eminences, giving to the whole an exceedingly irregular outline. In intra-abdominal growths manipulation usually shows that the tumors are movable from side to side. This is especially true of single pedunculated subperitoneal tumors. When the whole of the uterus is occupied by a myomatous mass, unless too large, it may be moved with some freedom. Intraligamentous growths are more restricted to one position. When the growth occupies both the pelvis and abdomen its pelvic portion holds it firmly in one position. Though inelastic and of firm resistance ordinarily, certain fibroids feel soft and even fluctuant. Rapidly growing myomata, especially when associated with pregnancy, are not as resistant as a tumor whose

growth is more gradual. With edema from any cause firm tumors may lose their resistance and yield a sense of elasticity or even fluctuation. Should myxomatous or any of the forms of degeneration befall the tumor, the entire substance within the capsule may be converted into a semi-fluid mass or true cyst, obliterating the characteristic fibroid feel, and yielding under palpation signs identical with ovarian cyst. Unless complicated by peritoneal inflammation, fibroids are not usually tender to abdominal palpation. Should ovarian cyst or pregnancy coexist with fibroma uteri, the palpating hand readily notices the difference in resistance between the fibrous and cystic growth; and while pregnancy greatly increases the vascularity of the tumor and softens it, it hasn't that elastic, yielding feel characteristic of the gravid uterus. Should the pregnant uterus lie surrounded by fibroids, abdominal palpation will throw but little light upon the diagnosis. The cases of pregnancy complicating fibroids especially easy of recognition are those in which the tumor lies distinctly laterally to the uterus and elevates that gravid organ out of the pelvis. The uterus and tumor may then be separately outlined, and the usual abdominal signs of pregnancy are easily elicited. In certain forms of myomata, especially those that are distinctly myomatous in structure, there have occasionally been felt alternate contractions and relaxations. Braxton Hicks, who should know something about this sign, says that the relaxation is never complete in myomata as in gravid uteri, and that its repetition is not so easily excited by manipulation.

By palpation and percussion we establish the disconnection of the tumor from the viscera. Fibroids are usually dull upon percussion over their whole surface, but more frequently perhaps than in ovarian cysts multinodular tumors of moderate size are covered by coils of intestine and yield areas of resonance upon percussion. When ascites complicates the presence of fibroma the diagnosis is not so easy. Trendelenburg posture will facilitate examination. Kelly refers to a pedunculated subserous tumor which, owing to the presence of ascites, yielded very distinct abdominal ballottement and closely resembled pregnancy.

Abdominal palpation is of value only in suprapelvic tumors; by it we may determine the size, density, shape, and multiplicity of the tumors. This knowledge is quite insufficient, it does not

satisfy us that the tumor is of uterine origin, nor does it in all instances show the relations of the tumor. But its chief deficiency is that it does not reveal the presence of intrapelvic low-lying tumors, which are oftentimes the most serious that we encounter.

We must, therefore, follow abdominal palpation by the combined vagino- and recto-abdominal examinations. It is impossible to describe all the phases revealed by these methods of examination. Polypoid tumors may lie in the vagina or be partially protruding from the os, or, lying within the uterine cavity, globularly distend the uterus. If of smaller size, they may be producing the most serious symptoms of hemorrhage and pain, yet concealed within the uterine cavity, inaccessible to palpation, and only found after dilatation and curettement. Tumors of the portio vaginalis and supravaginal cervix frequently have a downward evolution and, encroaching upon the vagina, preclude thorough digital exploration. As a rule, myoma of the body of the womb fixes the uterus in some position and blocks the pelvis more decidedly than cystic tumors. When the growth lies posteriorly to the uterus it is not infrequently sensitive, yet not so acutely so as inflammatory masses. It is of greatest importance to determine the position of the tumor with relation to the uterus, and this can be done usually by bimanual palpation. In mural growths, generally distributed throughout the uterus, the vaginal cervix feels like a hard knob attached to a denser mass. In such generalized growths we cannot outline separately the uterine body, it is all taken up in the tumor-formation. But when the tumor lies behind or in front of the uterus, or laterally to it, the experienced examiner can usually determine by combined palpation the relations of the part. The uninvolved portion of the uterus has a smooth, soft, and uniform feel, as compared with the nodular, harder, irregular fibroid. Even in soft myomata we recognize this distinction. The position of the portio vaginalis is often misleading as to the location of the body of the uterus. In pregnancy it is especially important that we ascertain the relations of the parts. An anterior fibroid may cause retrodisplacement and incarceration of the pregnant uterus, or a fibroid lying posterior to the pregnant uterus may, if left in that position, become incarcerated or attain to such dimensions as to preclude delivery.

Small tumors, high on the posterior surface or fundus, are especially liable to escape detection; but if the examination is conducted under anesthesia, as it always should be, and the patient is properly prepared, we may succeed in outlining these tumors by having an assistant draw the uterus well down with volsella forceps, while the examiner makes combined recto-abdominal palpation. With the uterus thus displaced, we get through the rectum a wider sweep and higher reach than we can through the vagina. Tumors that develop in the lower segment of the uterus may be capped, as it were, by the fundus. The external hand will here note the uterine softness as very different from the hard mass felt by the internal hand. Intraligamentary tumors are recognized by their low lateral position, their fixity, their relations to the vagina and rectum, and by the displacement of the uterus they occasion. I have often found myself in error, however, in attempting to make this diagnosis. Tumors of the fundus, especially the subserous pediculate, may draw the uterus out of the pelvis so that its cervix lies high and is difficult to reach. The essential object to be accomplished in the combined examination is to establish the attachment of the tumor to the uterus. This is done by ascertaining the blending of the two structures into each other or by finding the pedicle, and by demonstrating their joint mobility.

The use of the sound is scarcely necessary, yet the inexperienced are not to be condemned for its employment as a confirmatory test.

Auscultation: Auscultation is practically of negative value. In those exceptional cases of cavernous myomata a bruit may be heard. We should not fail, however, to auscultate the heart.

Differential Diagnosis.—**Ovarian cysts** may be mistaken for fibroids of the uterus. They rarely cause menorrhagia or metrorrhagia, and only amenorrhea from bilateral disease or through constitutional depravity. They do not occasion sterility, they are of more rapid growth, give rise to fewer pressure-symptoms, are more subject to inflammation, produce more symmetrical distention of the abdomen, and not infrequently cause enlargement of the abdominal veins; their surface is more uniformly smooth; they are soft, elastic, and fluctuating; they usually cause

descent and an antero- or retroposition of the uterus, and they are not intimately connected with this organ.

Pregnancy: Intramural fibroids may produce an abdominal tumor closely resembling a pregnant uterus. It is important to ascertain the history of the growth, its long duration, and the menstrual disorders that usually attend it. If these differential facts are wanting we must rely upon the physical signs of pregnancy, bearing in mind that myomatous disease of the uterus sometimes produces enlargement of the breasts and a flow of milk; that irregular uterine contractions may sometimes be felt; and that a venous hum may be heard. Fibroid disease never produces that soft, succulent cervix so characteristic of pregnancy. Pudendal veins may be enlarged from myoma, but the mucous membrane of the vagina and cervix does not show that blue cast seen in pregnancy. Internal ballottement and Hegar's sign are not simulated by any condition in fibroids. The detection of fetal parts by abdominal palpation, and the fetal heart-sounds should clear the diagnosis. If in spite of all these points one is still in doubt time should be called to his aid.

Tubal Disease: Chronic pelvic inflammation has not always a clear history of fever. In other symptoms it may closely resemble fibroids. It is chiefly by bimanual palpation that we discover the extremely sensitive, irregular swelling and are able to isolate the uterus.

Wandering Viscera: The *kidney* sometimes occupies a pelvic position, usually a congenital misplacement. Only occasionally does a movable kidney descend into the pelvis. The shape of the organ, the pain and nausea produced by manipulation, the absence of the organ from its normal position, would be our only means of escape from this plausible error. Hypertrophied *spleens* have in several instances been mistaken for uterine fibroids; I have myself made the error. Careful investigation as to the origin of the growth, the shape of the tumor, its thin notched border, the associate blood-conditions and dyscrasia, and the finding of the normal uterus displaced downward are sufficient means of differentiation.

PROGNOSIS.—By little we have become better acquainted with fibromyomatous disease of the uterus. We know that these tumors do not always occasion sterility. We have learned that the

menopause does not arrest their growth with any degree of certainty, but that, to the contrary, they defer the culmination of this period, and that, what is more lamentable, not infrequently these tumors become pathologically active only after the menopause. Pregnancy, as is shown, stimulates their growth, and occasions the accidents of infection, torsion of the pedicle, and necrosis. Labor has its dangers, and the parturient state may be seriously affected by the presence of a fibroid. We are only lately recognizing that tubal and ovarian disease is a most uncommon complication. And as yet the profession is not sufficiently alive to the complications affecting the heart and kidneys; nor does it realize that a central necrosis in a subperitoneal fibroid may supply intoxicating elements. Cystic and malignant degenerations may befall a tumor at any time in its life-history. With this rather dismal but true picture before us, can we subscribe to the statement of Champneys that the mortality from fibroids apart from operation is practically nil? Dark as the picture of fibroids with their complications appears, truth compels the admission that many tumors may behave in the most innocent and inoffensive manner; but my experience teaches that when a fibroid symptomatically asserts itself the woman's life is practically marred. The optimistic view held by the English that the great majority of these tumors do not require operative interference is, upon authority of one of their most eminent countrymen, Mr. Hart, somewhat biased because of the unfortunate remote results that attend the operation of extraperitoneal treatment of the stump with the serrenoeud, and because of a rather exaggerated idea of the beneficial results occurring at the menopause.

The medicinal treatment of fibroids, electrolysis, ligation of the uterine arteries, vigorous curettage, and castration are measures which are at most palliative, uncertain in their results, not without dangers of their own which approximate those incurred in more radical measures, and are, one and all, becoming obsolete.

The results of the operative treatment constitute the most brilliant achievement of surgery in the last half-century. In this country, considering the number of cases treated, Kelly's record is unsurpassed. Between 1894 and 1900, he has operated upon

307 myomatous uteri with a mortality of 4.88 per cent. In 433 cases operated upon in the London hospitals by various surgeons the average mortality was 17 per cent. (Champneys, 1900). Theinhaus cited before the Gynecological Congress, at Berlin, 1900, 564 operations of hysterectomy and morcellation at the hands of nine different surgeons with a total mortality of 2.3 per cent. Ricard's statistics, reported in 1899, of 2290 hysterectomies show a mortality of 7.075 per cent. These brilliant results represent the work of the masters. I dare not suggest what the real mortality is, could an average be taken of the results of all who presume to do this work. For myself, I cannot claim a mortality, when all cases are included, of less than 7 per cent. The immediate mortality of hysterectomy is usually due to shock from prolonged anesthesia and hemorrhage, secondary hemorrhage, infection, cardiac failure with brown atrophy, intestinal obstruction, and to pulmonary embolism. Convalescence is complicated by pelvic suppuration, due to infection in the stump; the constricted tissues necrose and with their ligatures are discharged, after having caused much local disturbance and not infrequently chronic sepsis. The precipitated menopause may bring about the most unpleasant phenomena.

Myomectomy, in the hands of a few, is regarded a safer procedure than hysterectomy. In properly selected cases this will prove to be the rule. That it can become the most popular operation seems most unreasonable. There is always danger of secondary hemorrhage and infection, and no conscientious surgeon can declare that he has removed all the tumors; many cases have required second operations; Martin and Kelly have both had such experience, while Doléris reports four cases of myomectomy in which small fibrous nodules, regarded as insignificant at the time of operation, subsequently developed to such extent as to require removal. *It is now a settled question, that fibroids should be treated surgically by one of several methods. We have yet to give the proper study to the remote results of the special operations.*

CONTRAINDICATIONS TO OPERATION.—A small fibroid that is not producing symptoms should be left alone. Subserous tumors, hard, of slow growth, of moderate size, should be watched. If the patient is nearing the menopause, their pathological history may cease at this crisis. Mural tumors that are quiescent, not

causing serious symptoms, may undergo retrogression. In our study of the coexistence of fibroids and pregnancy we have set forth the contraindications to and indications for interference. Varicosities of the veins of the pelvis and extremities, while not an absolute contraindication, is a complication for serious consideration. Cardiac and renal disease, unless too far advanced, is an indication for, rather than against operative interference. Marked anemia may render intervention unjustifiable, yet Da-Costa reports a successful case of pan-hysterectomy in which there was only 21 per cent. of hemoglobin. In patients with less than 40 per cent. of hemoglobin the danger is always increased, but this should not deter us from operation.

INDICATIONS FOR OPERATION.—Functional disorders, irrespective of the size of the tumor and age of the patient, may demand operation. All those conditions arising from the tumor or its complications, which imperil the life of the patient, call for operation. Hemorrhage, pressure-symptoms, incarceration, infection, disease of the appendages, torsions of the pedicle, cystic or malignant changes, are all conditions for immediate interference. Life may not be imperiled by the behavior of the tumor or the development of complications, yet the subjective symptoms, the burdensome existence, demand relief.

SURGICAL INDICATIONS.—Polypoid growths are removed through the vagina. Submucous tumors of sessile attachment and of moderate size, if capable of being pressed into the pelvis, should be attacked through the vagina. Doyen's improvement of slitting up the anterior uterine wall and *morcellement* by spiral incision favor this procedure. The enucleation of submucous fibroids is a very tedious and exhausting procedure, and I have sometimes regretted undertaking this work.

Vaginal hysterectomy should be restricted to those tumors which lie in the pelvis. It is questionable if this procedure possesses any advantages over the abdominal route, and its successful performance is contingent upon so many conditions that it will never become the universal method.

Hysteromyomectomy, either partial or complete, with preservation of the ovaries when possible, is the most universally employed procedure.

UTERUS.

SUBSEROUS SESSILE AND PEDUNCULATED FIBROMYOMATA OF THE UTERUS.

Supravaginal amputation after the method of Baer, modified by Kelly, Pryor, Hall, and others, has a mortality of practically 5 per cent.; these results are obtained by many operators. It has the advantage of being generally applicable, requires but a short time for its execution, hemostasis is perfect, there is no needless exposure of subperitoneal tissues, we can safeguard the ureters, and the anatomical relations of the vaginal vault are preserved. The apprehension of malignant degeneration of the cervical stump is chimerical. Even when adenocarcinoma of the body of the uterus complicates fibroids, it shows but little tendency to invade the cervix. Still, if this diagnosis is made prior to operation, removal should be complete. The objections to this operation are: first, a question of technic—when the uterine arteries are isolated and tied, we are taught by some that it is not necessary to tie the veins; this omission favors the formation of thrombi and emboli, and postoperative vomiting and coughing force the blood from the veins, favoring the development of sapremia; Zweifel considers the ligation of these valveless veins indispensable. Second, another source of hemorrhage, perhaps unnoticed at the time of operation, is from the raw surface of the amputated cervix; to avoid this, these raw surfaces of the stump should be brought together with catgut sutures. Third, necrosis of the stump, with mild sepsis, purulent discharges and escaping ligatures, are frequent complications that prolong convalescence. For intraligamentous fibroids the technics of Hall, and Kelly render bloodless and simple what would otherwise be always a difficult and sometimes an impossible procedure.

Pan-hysterectomy should be chosen in all cases showing infection, and in those in which malignancy is suspected. This operation is growing in popularity. There is better drainage, local infections from necrosed tissue is avoided, the operation consumes but a few minutes more time, yet for some reason the mortality is somewhat greater than that of supravaginal amputation.

Myomectomy is unquestionably the ideal operation. It removes the visible and palpable disease, leaves the uterus and its appendages capable of performing their functions. It does not insure against the development of undetected fibroids, whose growth may be actively progressing. It is the desirable operation in young women, and is specially applicable for the removal

of pedunculated and sessile subperitoneal fibroids, multiple or single, and of interstitial tumors of moderate size and well-encapsuled. Martin extends its application to large submucous tumors which cannot be removed through the cervix. The safety of the operation depends largely upon the thoroughness with which the bed of the tumor is closed with buried catgut sutures.

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NEOPLASMS OF THE OVARIES AND BROAD LIGAMENTS.

In view of the many unsettled problems of their histogenesis, a simple clinical classification of ovarian neoplasms is most tenable.

- I. Benign Cystic Neoplasms.
  - A. Ovarian
    - (a) Simple Hydrops Folliculi.
    - (b) Follicular Degeneration of the Ovary.
    - (c) Cystic Corpus Luteum.
    - (d) Cystadenomata
      - (1) Pseudomucinosum.
      - (2) Serosum.
  - B. Intraligamentous.
  - C. Dermoid.
- II. Benign Solid Neoplasms.
  - Fibroma and Fibromyoma.
- III. Malignant Neoplasms.
  - (1) Carcinoma.
  - (2) Sarcoma.
  - (3) Endothelioma.

I. Benign Cystic Neoplasms.

A. Ovarian Cysts.—(a) **Simple Hydrops Folliculi:** When from insufficient bursting force, from thickening of the tunica albuginea due to cirrhosis or to previous inflammation, or from causes unknown the physiological cycle of ovulation is arrested with the follicular wall intact, there accumulates a fluid, the product of the epithelial lining, the retention of which constitutes dropsy of the follicle. It is difficult to determine when this process becomes pathological; they doubtless frequently form and harmlessly rupture, and so disappear. When, however, the cyst-wall is sufficiently resisting to permit the accumulation of some ounces or more of fluid they become of surgical importance, yet one should be very guarded in pronouncing a unilocular cyst of the ovary of large size follicular hydrops. But the occurrence of these simple unilocular cysts springing from the oöphoron, containing serous fluid, sometimes mucoid in character and blood-stained, in which fluid the ovule cannot infrequently be demonstrated, with a sac-wall lined with a membrana granulosa, establishes their

follicular genesis. A follicular cyst bulges from the free surface of the ovary without alteration in the parenchyma. These simple retention-cyts, when single, may attain to the size of one's fist or even to that of a child's head; in such cysts the cylindrical epithelium is flattened or effaced by atrophy, and the ovule has disappeared or escapes detection. Several follicles may be simultaneously affected, and the condition may appear in both ovaries.

(b) **Follicular Degeneration of the Ovary (Simple Multilocular Cyst)** In this condition the ovary consists of numberless follicles which have ripened and become dropsical, each separate cyst possessing the individual characteristics of a follicle. They project beyond the surface of the ovary, and through their thin walls the clear contents are visible. Each cyst may be no larger than a pigeon's egg, though the conglomerate mass of the degenerated ovary may form a bulky tumor. When the cysts enlarge they may coalesce so that there may be one cyst of rather large size with many smaller ones. Many authorities hold that this is the incipient stage of the cystadenoma, but Schultz, and Gebhard maintain that the process begins in the follicle itself. Many views are expressed as to the cause of simultaneous multiple degeneration of the follicular structure. It is impossible to deny the pathogenetic effect of inflammation, of long continued congestion, venous stasis, of primary degenerative diseases of the blood-vessels; but Schultz offers a plausible theory when he ascribes it to irritation of the ovarian nerves, either reflex or direct, leading to the simultaneous maturation of many follicles. Whatever their origin, there is no proliferation of epithelium or formation of new cysts, in which respect they differ from true cystadenomata.

(c) **Cystic Corpus Luteum:** A cyst may develop from a collection of serous fluid in a corpus luteum, and like follicular cysts it is usually of small size, develops slowly, and generally exists as a solitary cyst. They are differentiated from follicular cysts in that they have thicker walls, in the inner stratum of which can be demonstrated the yellow granular sheath of luteum-cells, though recently Fränkel has shown that such cysts may have an epithelial lining.

(d) **Cystadenoma:** When the world's greatest pathologists, after the most painstaking labor, arrive at entirely different con-

clusions as to the histogenesis of ovarian neoplasms, it is the height of presumption for practical surgeons to write dogmatically upon the subject. Whether the compound proliferating ovarian cystoma originates from the Graafian follicle, as is held by the highest English authorities, Tait, Sutton, and Doran, or whether the view of Waldeyer, who assigns their origin to the germinal epithelium, a theory that is accepted by Germans and by the majority of American writers with the exception, perhaps, of Coe, is correct, remains an open question. Personally I find it difficult to reject the position taken by Mr. Doran, and thus stated: "The most probable origin of cystic disease of the ovary is an arrest of the normal retrograde metamorphosis of the Graafian follicles that have never become corpora lutea of menstruation or pregnancy." The parenchyma of the ovary is formed of the germinal epithelium and its legitimate offspring, the follicular epithelia and the ovules. From each of these structures epithelial neoplasms, both cystic and solid, may arise. Their growth is due to the multiplication of the glandular elements of the ovary. The arrangement of the cells determines their benignity or malignancy.

In the development of adenomata there is a never-failing tendency to cystic formation, and such tumors of the ovary are designated as cystadenomata. Sutton was the first to ascribe the glandular character to the multilocular oöphoritic cyst. Waldeyer divides the proliferating cysts into two groups: the proliferating glandular, and the proliferating papillary cysts. Olshausen, and later Williams, and Pfannenstiel draw quite a sharp distinction between the glandular and papillary varieties. A simple and satisfactory division of cystadenomata is that of Pfannenstiel into (1) those containing pseudomucin, and (2) those with serous contents.

1. *Cystadenoma Pseudomucinosum (Multilocular Oöphoritic Cyst):* This is the most common of all varieties of ovarian tumors. Out of 605 cases of abdominal section for all conditions, 366 were for the removal of these growths (Doran). But little is known as to the cause of these tumors. The predisposing age is between twenty and forty years, the period of sexual activity, and they are more common in the unmarried and sterile. It cannot be said that there is any inherited tendency, yet strange coincidences of

their occurrence in several individuals in the same family are recorded; Löhlein refers to a family history in which bilateral ovarian cystomata occurred in three sisters. Multilocular ovarian cysts are infrequent in the negro; dermoid and parovarian cysts are more common.

These multilocular adenomatous cysts often grow rapidly and to enormous proportions; Cordier removed one weighing 160 pounds; in Cartledge's case the cyst and its contents weighed 245 pounds. As they spring from the periphery of the ovary, the oöphoron, their shape depends largely upon their size and location. A tumor the size of a child's head is usually smooth or ovoid, occasionally nodular, with an elastic feel. When of small size they lie in the pelvis behind the uterus, pedicled to it by the tube, ovarian ligament, mesovarium and mesosalpinx. Larger cysts rise into the abdomen, displace the intestines to the side, adapt themselves to the concavities, and rest in close apposition to the parietes. Upon exposure by abdominal section the main cyst-wall appears smooth, glistening, and silvery-white. The bosses, that is, the secondary cysts, the walls of which are thin, are either pearly-white, of a bluish, opalescent tint, or pink, or sometimes dark-brown from discoloration by blood. The entire ovary is consumed in the tumor-formation. Usually there is one large cyst enclosing numerous other cysts of all sizes, some bulging from the parent cyst, others projecting into its cavity. On opening the tumor it is seen to consist of several closed cyst-spaces, the whole forming a more or less extensive conglomerate mass enclosed in a capsule of connective tissue. In the formative stage of a cystadenoma there is irregular development of the follicle with active changes in its cylindrical epithelial lining. Tube-like diverticula from the cyst-wall project into other parts. The rapid cell-proliferation leads to an accumulation of a tough secretion in these tubular spaces. These ampulla-like distentions become endogenous or daughter-cysts, which in turn proliferate; and by a repetition of this process innumerable cyst-formations are produced. These secondary cysts may rupture the parent cyst-wall and appear as bosses or false exogenous cysts. They are characterized by their thin walls and bluish lustre. True exogenous cysts may form independently, that is, never connected with the main cyst-wall. The tendency of secondary cysts is

to open into each other and into the main cyst by pressure-atrophy, thin trabeculæ of fibrous ridges indicating the site of septa. In old tumors all traces of the original multilocular state may have disappeared. The fusion of coexisting cysts of each ovary may result in a large cyst with a double pedicle; Doran has met with five such instances.

The normal ovary lies free in the abdominal cavity, its greater portion being covered by its germinal epithelium and a small part by its peritoneal lamella. Tumors of extraligamentary evolution retain the same relations as the normal ovary.

These growths are usually unilateral, though not infrequently a similar process may affect the other ovary. When not involved in cystic degeneration the other organ may be found atrophied or in a state of passive congestion from pressure. These cysts are not infrequently found combined with dermoid cysts in the same or in the other ovary.

The cyst-wall histologically consists of three layers: the external is endothelial in character. The middle layer abounds chiefly in white fibrous tissue, and near the pedicle, especially on the inner side, plain muscular fibers are present; in this middle layer are distributed the blood-vessels, the capillaries ramifying in the subendothelial stratum; the veins are often large and lie very superficially; nerves and lymphatics ramify throughout the walls, the lymphatics being especially demonstrable near the pedicle; secondary cysts are sometimes found in this coat of the wall, and occasionally corpora lutea may be demonstrated. The inner lining or third coat of the cyst-wall is vascular unless too greatly thinned by distention, and in small cysts, according to Sutton and to Doran, it is separated from the middle layer by a true submucous tissue, contains lymphoid elements, and is covered by columnar epithelium; in larger cysts the intrinsic pressure has flattened the cells, and their nuclei then rest upon the basement-membrane; tall goblet-cells are frequently found lining some of the secondary cyst-cavities, especially those with colloid contents; the internal surface is usually smooth, yet ingrowths of epithelium are frequently noticed.

While such are the characteristics of the cyst-wall of an unmixed pseudomucinous cystadenoma, papillary masses and dermal elements may be found, which add to the complexity of

the tumor. The external appearance of the cyst is influenced by so many accidental conditions which affect either the cyst-contents or its walls that one must be prepared for the greatest variations in the classical picture. Mural hemorrhages destroy the lustre, causing yellow or brownish areas. Calcareous deposits harden the walls. The accidents of inflammation, torsion of the pedicle, and intracystic hemorrhages are responsible for very deceptive conditions.

The contents of a glandular cystoma vary in consistency usually with the size of the tumor. In smaller cysts the fluid is often colloid, thick, elastic, and tenacious. In larger cysts the fluid is generally thinner; in other cases it is a colloid material; it varies in color from a milk-white to yellowish-brown, green or red; it sometimes looks like boiled starch; it varies in specific gravity from 1010 to 1030, is non-coagulable, and is chiefly the product of epithelial activity, with some admixture of blood-serum. Not infrequently it is beautifully colored from the admixture with blood. Chemically it is designated by Hammarstein and by Pfannenstiel as pseudomucin. Cysts with colloid contents not infrequently have very thin walls, and are liable to rupture, the escaping fluid setting up a pseudomyxomatous peritonitis. Cholesterin imparts a peculiar greenish shimmer to the contents of ovarian cysts. Microscopically epithelial cells, fat and numerous granules, epithelial scales, blood-corpuscles, and cholesterin-crystals are found.

The multilocular cystadenomata, developing usually from the free surface or parenchymatous structure of the ovary, lie intraperitoneally and are therefore usually pediculate. If of small size the pedicle consists of the ovarian ligament and that portion of the broad ligament known as the mesosalpinx and mesovarium. In tumors of larger size with increased weight and traction, the Fallopian tube is generally drawn up alongside the tumor, the outer portion of the broad ligament, the infundibulopelvic ligament, with its contained vessels and nerves, is drawn inward, so that the pedicle usually consists of the following structures: the upper fold of the broad ligament containing the Fallopian tube, the terminal portion of the uterine artery and vein along the inner border, the ovarian artery and the vein on the outer border, embraced in the infundibulopelvic ligament, the

ovarian ligament, and not infrequently some portion of the parovarium, and generally in its central portion a mass of dilated veins from the pampiniform plexus. The pedicle may vary in length from one to several inches; its usual length is about two inches. It is of variable dimensions, from a slender cord to a broad fleshy band; in some instances it is a merely membranous connection. Broad pedicles are usually quite short.

When the cyst develops from the medullary strands in the hilum, that is, in the proximal portion of the ovary, the tendency of growth will be intraligamentary. It grows into the loose connective tissue of the mesovarium and opens up the broad ligament. Such a tumor is not only covered by its own capsule of connective tissue, but by the component parts of the parametrium. It is sessile in attachment, and is recognized by its detachable, vascular covering and its relation to the Fallopian tube. Usually the tube lies directly over the tumor, and this oviduct is greatly lengthened. A cyst may be both intraligamentary and abdominal in development. We must not mistake a tumor which pushes the broad ligament before it and over it for a true intraligamentary cyst. In one instance the tumor lies between the folds of the broad ligament; in the other, it is behind the broad ligament and is covered by both its folds of peritoneum.

2. *Cystadenoma Serosum* (*Papillomatous Paroöphoritic Cyst*): In designating these papillomatous cysts as paroöphoritic cysts it is not intended to restrict their origin to the hilum of the ovary, as is taught by Mr. Doran, but it is the purpose rather to group the synonyms by which cysts containing epithelial masses are variously known. In further explanation, under this heading we shall consider surface non-cystic papillomatous tumors of the ovary, since they in no way differ histogenetically from those attended by cystic formation.

These tumors are proliferating adenomata with serum-like contents, and contain papillary epithelial growths. Pseudomucin is not found in their fluid contents, thus grossly distinguishing them from the ordinary cystadenoma pseudomucinosum. Comparatively speaking, they are infrequent forms of ovarian tumors; their frequency is variously estimated at from 8 to 15 per cent. In 1200 abdominal sections for all conditions Kelly found 31 papillomata. Doran, whose observations were based chiefly upon

the immense work at the Samaritan Hospital, at the time of his writing upon this subject had only seen 24 cases. The cystic papillomata are more common than the surface ones; only 4 of Pfannenstiel's 60 cases were of the exclusively surface sort. While this form of growth is occasionally met with in patients under thirty years, these papillary tumors usually appear late in life; Doran found one instance in a seven-months' fetus. Their growth is slow during the first part of their history, but when ascites and metastasis develop the case generally pursues a rapid clinical course. Another characteristic is their bilateral tendency. Papillomatous cysts are usually of small size, rarely attaining to the size of an orange or of a cocoa-nut. Their intraligamentary location may give rise to pressure-symptoms. Pain is especially experienced during the clinical history of these cases when the cysts rupture, or in surface papillomata that choke the pelvis. Whatever their origin may be, there is a marked tendency to intraligamentary development. They are rarely pediculate, and the ovary often retains its form.

The cyst-wall is of delicate texture, and spontaneous rupture is of common occurrence, followed by papillomatous implantation on the peritoneal surface; and this tendency to metastatic formations in the parietal and visceral layers of the peritoneum constitutes one of the chief characteristics of the growth. Ascites is present in more than one-half the cases. Abundant free fluid is especially observed in those cases in which the papillary masses project into the peritoneal cavity. The ascitic fluid is, no doubt, the product of the tumor, may be turbid, straw-colored, or red from the admixture with blood. The appearance of secondary papillomatous growths and hydroperitoneum are considered by many as evidences of malignancy. It is difficult, even impossible, to differentiate microscopically the glandular papillomata from an adenocarcinoma; the mere evidence of peritoneal metastases does not imply a necessary reoccurrence; after removal of the tumor the secondary deposits often disappear.

The cysts are usually multilocular, and on section there will be found springing from the inner lining pedunculated or sessile dendritic masses of papillary excrescences, consisting of connective tissue covered by columnar ciliated epithelium; they often grow very luxuriantly, forming cauliflower-like masses, filling

the cyst, penetrating its walls, engrafting themselves chiefly upon the peritoneum of the pelvis, enveloping and concealing the ovaries and tubes. The villous processes are often hard and gritty, containing psammomata which are probably due to degeneration of the epithelial structures. They are often richly supplied with blood-vessels, as is evidenced sometimes by their pinkish color and marked tendency to bleed upon rough handling. Hyaline degeneration of these blood-vessels seems to be a constant accompaniment. The secondary growths show a great tendency to cystic formation.

The contents of the cyst is often clear, straw-colored, or bloody, rich in albumin, but does not contain pseudomucin.

Olshausen believes that the papillary cystadenoma develops from the parovarium because it contains ciliated epithelium. Doran long ago cautioned against placing too much stress upon the presence of ciliated epithelium; he thinks they arise from Wolffian elements that have penetrated the hilum and parenchyma of the ovary. Uffenheimer regards the appearance of cilia as merely the result of metaplasia. Pfannenstiel has shown that cilia may be formed from the germinal epithelium. Orth says that they may develop from the germinal epithelium, from the follicle-epithelium, or from the medullary strands. With this view Williams accords, though he considers that the papillary cyst usually develops from the Graafian follicle, and will be lined with ciliated or non-ciliated epithelium according to the condition of the cells of the membrana granulosa: surface papilloma is most frequently of germinal-epithelium origin.

B. Intraligamentous Cysts.—Reference has already been made to the occasional intraligamentary development of ordinary cystadenomata of the oöphoron. The papillary cyst of the hilum frequently develops between the folds of the broad ligament. But the true broad-ligament cyst is of parovarian origin, that is, it springs from the tubules of the parovarium, the organ of Rosenmüller, which is the tubular embryonic remains of the Wolffian body, or from the ducts of Gärtner. These tumors are always primarily intraligamentous; others may become so secondarily. One in every six cysts developing in the ovary and its vicinity are intraligamentous.

Such cysts may, in their development, become pedunculated but only when they grow into the abdomen, when they are liable to the accident of axial rotation. If their evolution is downward, separating the ligament and elevating the parietal peritoneum, they remain sessile. They may develop in both directions, and often attain to great capacity. The cysts are unilocular, rarely contain papillomatous growths. Hirst reports a case of coincident ovarian and parovarian cyst in the same person, the only such instance we find recorded.

Their fluid contents is clear, of low specific gravity, 1002 to 1004. Their walls are thin. The inner surface is corrugated, lined with cubical, sometimes cylindrical epithelium, and may contain papillary excrescences. The outer surface is smooth and glistening, to which the peritoneum is loosely attached, and smooth muscular fibers are seen passing over the outer wall. The blood-vessels of the peritoneum have a direction differing from those of the tumor, so that the two well-defined vascular net-works are seen crossing each other (Kelly). The Fallopian tube passes over the top, anterior or posterior surface of the tumor, and bends around toward the ovary. The tube is elongated according to the size of the tumor, its fimbriæ stretched, but otherwise it is normal. The ovary flattened, atrophied or normal, lies on the lower posterior surface, as a rule. In those tumors of abdominal development the surface is smooth and regular, but if the growth is downward between the folds of the ligament, the tumor is constricted in parts and irregular in outline. The sac is often flaccid, not tense, like in oöphoritic cysts. Large cysts of intra-abdominal development, owing to their thin walls, may spontaneously rupture; their escaped contents is innoxious to the peritoneum.

Small pedunculated cysts of Kobelt's tubes and other subserous collections, frequently seen beneath the broad ligament, are unimportant since they rarely become large.

C. Dermoid Cysts.—A mixed heterologous cyst of the ovary of congenital origin, the contents consisting chiefly of dermal elements, hence its name. While ectodermic derivations predominate, tissues from all three layers of the blastoderm may be found.

No period of life is exempt from their occurrence. Olshausen refers to three instances in premature infants and to several cases

in childhood, and an ovarian dermoid has been found in a woman eighty-four years of age. The majority of these cysts develop soon after puberty and during active sexual life, stimulated by the physiological energy of the ovary. Their relative frequency to other ovarian tumors is placed at from 3 per cent. to 24 per cent. by various authors. In Kelly's table they constituted 18.84 per cent. of all cystic neoplasms of the ovary. In 411 cases Novy found 42, about 10.2 per cent. Authorities generally hold that they occur most often in girls and sterile women. Pately's statistics of 118 cases show 49 unmarried, and 69 married, of whom 5 were childless. Of the author's 9 cases 2 were married, 7 unmarried, the ages ranging from 12 to 30 years. In the majority of cases it appears that the right ovary is affected, but this must be a mere statistical accident. Both ovaries may be simultaneously the seat of dermoid changes, or there may be a dermoid of one and a neoplasm of another sort of the other. Dermoids are frequently combined with the cystadenomata. Unless combined with proliferating cysts, dermoids are usually unilocular and single, yet Wilms describes a case in which there were 5 dermoid cysts present.

Dermoid cysts of this region do not always take their origin from the ovary. They may be found in the broad ligament, the ovary taking no part in their formation. Bertholet, in 1898, collected 9 published cases of dermoids in the ligamentum latum free from ovarian attachment, which is evidence rather conclusive of their origin from misplaced cells of the Wolffian body.

Tumors of this sort are of moderate size. They may be no larger than a hen's egg and rarely attain to a growth larger than a child's head, though they have been found to weigh more than one hundred pounds (Keith). They are proportionately heavier than other cystic tumors because of the nature of their contents. Ovarian dermoids usually have long pedicles, and are especially liable to the accident of axial rotation. They are also liable to infection; consequently in their clinical course they often excite attacks of peritonitis and are frequently densely adherent. Their growth is usually attended by greater pain and more pronounced menstrual disorders than in cases of other benign cysts. Rupture of the cyst-wall occasionally occurs, usually from sudden distention, the result of torsion of the pedicle. A fatal peritonitis

is the usual sequence. As in the proliferating papillary cyst, minute dermoid growths have been found disseminated over the peritoneal surfaces. There is no good reason, says Sutton, to doubt the occurrence of transplantation in dermoids. Of more consequence is the tendency to carcinomatous and sarcomatous degeneration. Clark refers to several cases of carcinoma developing from the epidermal structure. Yamagiva has reported a unique case of glandular carcinoma developing from a misplaced mammary gland on the inner surface of a large dermoid cyst.

On exposure a dermoid may appear very differently under varying conditions. It rarely has the glistening, shiny appearance of more simple cysts. When small, inflamed, and covered by adhesions, both in appearance and by feel it may resemble a uterine fibroid. It is often dark, of a mottled brown appearance, and when the subject of torsion of the pedicle it may be dark-green or even black. The walls, though sometimes quite thin, are usually much thicker than those of other cysts. The inner lining of the sac, which corresponds to the epidermis, consists of layers of epithelial cells upon a connective-tissue basis, beneath which there is the panniculus adiposus. Sometimes the entire cyst is so lined; more frequently there are only patches of dermal structure.

Every variety of tissue has been demonstrated in dermoids; hair, sebaceous glands, sweat glands, teeth, embedded in the wall or implanted in a rudimentary maxilla, bone, nails, unstriped muscle-fibers frequently, and occasionally striped muscle, and tissue resembling brain-matter. Fat, as pultaceous grease, often in pill-like masses, each pellet containing a hair, has been found to fill these cysts. The fluid contents is usually of a characteristic greasy look, of chocolate color, which, if exposed to the air, will form upon its surface beautiful hexagonal patterns of cholesterin crystals.

The most universally accepted theory of the histogenesis of dermoid cysts is that of Cohnheim, which ascribes the origin of these tumors to the nipping off or inclusion of a portion of the ectodermal layer in the early growth of the embryo. These misplaced skin-elements, stimulated by ovarian congestion, take on atypical growth. Wilms first offered substantial proof of the ovulogenous origin of dermoids, a suggestion plausible and ac-

ceptable, inasmuch as it is known that the ovum possesses the germs of all tissues. Krömer considers dermoid cysts to be of both follicular and ovulogenous origin, the cystic portion supplied by the follicle and the tissue-elements by the ovule. Clark, in reviewing Krömer's work exhaustively in Progressive Medicine, says: "Until very strong evidence is brought in opposition to Krömer's excellent studies we must put strong credence in his theories." This whole question is hypothetical.

COMPLICATIONS.—**Infection:** An ovarian cystoma is a true neoplasm. In its uncomplicated development it is not attended by inflammation, and the sac and contents are sterile of pathogenic microörganisms. By its presence it may so mechanically irritate the peritoneum as to produce a plastic exudate, resulting in adhesions; but ordinarily extensive adhesions are evidences of infection. More serious invasion may result in ulceration and gangrene of the sac. When the contents of the cyst is contaminated the fluid becomes purulent in character. Constitutional conditions impairing the general health are predisposing causes of infection. Any traumatism or circumstance impairing the circulation in the cyst-wall invites infection. Constriction of the pedicle from torsion or otherwise renders the sac and its contents especially vulnerable. Incarceration will act in a similar manner. Hemorrhage from injury or other cause may be the initial step in suppuration of the cyst. The pregnant uterus displaces and compresses the sac, rendering it more liable to microbic invasion. Labor, normal or instrumental, may result in contusion and injury of the sac or even rupture. Papillary and malignant growths, because of their tendency to intracystic hemorrhage, are especially prone to infection. Small tumors impacted in the pelvis, because of their relation to the rectum, suffer infection very readily. Therefore, on account of their size, position, and slow growth, dermoid tumors are more liable to this accident than tumors of less density and more rapid growth. Mangold claims, though it is not established, that spontaneous suppuration of dermoids may occur from the setting free of pyogenic irritating substances from the cyst's contents.

When tapping was in vogue it was a common mode of conveying infection to the sac; fortunately but few patients are subjected to this danger now. The most usual channel of invasion

by microörganisms is through the lymphatics, as infection of ovarian cystoma in a puerpera; Zetter reports 21 cases occurring during the puerperium. Infection from the intestines is always a gradual process; the sac and bowel are mutually impaired by compression, and the intestinal organisms penetrate the tissues, establish a localized peritonitis with adhesions and thickening of the cyst-wall, and may ultimately contaminate the contents. A suppuration under these circumstances is attended by offensive odor, and not infrequently by formation of gas. Very active inflammation may occur from an adherent infected appendix or Fallopian tube. Cryptogenic infection is said to occur, and suppuration of the cyst's contents from the bacillus typhosus is cited in proof. While admitting this mode of infection we must also recognize that it is a peculiarity of this special bacillus to remain latent for some time in its points of lodgment; and it is possible that in several of the instances of suppuration of ovarian cysts during and following typhoid fever the cyst-wall was in contact with the intestinal lesions, and the mode of conveyance was through the lymphatics. Werth, Pitha, and Mangold record instances of pure cultures of Eberth's bacillus obtained from the pus of suppurating cystoma. It is more probably the blood that was the medium of infection in Schauta's case of suppuration due to the diplococcus pneumoniæ.

Mangold, and Cumston hold that infection may be either from the ordinary pyogenic microörganisms or from saprophytes. In puerperal infection streptococci and staphylocci have been repeatedly demonstrated. In infection from the alimentary canal the bacillus coli communis is conspicuous. The bacillus typhosus, diplococcus pneumoniæ, gonococcus, and bacillus tuberculosis have all been found.

The milder grades of infection, localized and limited to the cyst-wall, cannot be recognized. In more pronounced instances the patient suffers from transient attacks of localized peritonitis. In chronic infections resulting in thickening of the wall and suppuration of the cyst's contents, the abdominal discomfort becomes more distressing, the general health is greatly impaired, a hectic temperature is recorded, and the patient finally develops a diarrhea and succumbs to exhaustion from chronic sepsis. When virulent infection, especially the streptococcus form com-

plicating the puerperium, takes place, we have all the symptoms of acute peritoneal inflammation with onset of chill and high temperature, and rapid increase in the size and tension of the tumor, tenderness especially marked over the tumor, with rapidly developing tympany and not infrequently peritoneal effusion.

In these cases of puerperal infection of ovarian cysts Zetter places the maternal mortality at 30 per cent., others as high as 50 per cent. The prognosis will depend largely upon the time of infection with relation to labor; the sooner it occurs after labor, the more hazardous the necessary operative procedure.

Torsion or Axial Rotation of the Ovarian Pedicle: The rotation of the tumor about its pedicle is the most frequent serious complication occurring in the life-history of ovarian cystoma. It is met with in from 6 per cent. to 30 per cent. of all cases. Olshausen places the frequency at 6.3 per cent., Küstner at 38.8 per cent. The author has met with it in fully 10 per cent. of his cases. It occurs with equal frequency in tumors of either side. It is especially frequent when ovarian cystoma and pregnancy coexist. In the only two instances in which I have been called upon to operate for cystoma during pregnancy, torsion of the pedicle precipitated the emergency. Kelly states that spherical cysts are peculiarly liable to an accidental rotation. The reverse is my observation. Thornton, and Doran both hold that tumors of irregular form are more liable to rotation. This is especially true when the long axis of the tumor lies transversely. The pedicle may be either long and slender, or short; it does not appear that its length has any special bearing upon the tendency to rotation. In case of bilateral tumors of unequal size the smaller is more prone to torsion. Dermoids, because of their size, irregularities, and asymmetry in weight, meet all the predisposing requirements and are very frequently rotated.

The active causes of rotation is a question concerning which there are many theories. The impaction of fecal matter, peristaltic action of the intestines, the alternate filling and emptying of the bladder, changes of posture, the action of the abdominal muscles, the contraction of the platysma muscle on the inner side of the pedicle, displacement of the tumor due to pregnancy, are factors acting singly or in combination to produce rotation.

It is impossible to attribute to any one cause a constancy of action.

Herman Freund's law of pedicle-torsion, based upon the old theory, marked out by Goodsir and Fischer, that all organisms grow in a spiral direction, applied to ovarian tumors, shows that tumors of the left side have a dextrospiral twist, and those of the right a sinistrospiral twist of the pedicle. Küstner found this to be a fairly constant rule.

The complication of torsion may be bilateral with different degrees of acuteness. In one side the rotation may gradually evolve, and with the supervention of some precipitating cause the opposite side may become suddenly constricted; Doran reports such a case. The effect of torsion of the pedicle is to cause nutritive changes in the tumor. The inner border of the pedicle is thick, resisting, and contains the larger arterial supply; the outer thin, soft, and conducts the veins and ovarian artery. The twisting force may be sufficient to arrest the venous circulation, to lessen but not obstruct the arterial supply; a venous congestion follows, resulting in dilatation of the veins, thickening of the sac, extravasations of blood into the walls of the sac, and often copious hemorrhage into the cyst. In some cases thrombosis of the veins will be observed. The peritoneal covering is eroded, becomes deep red, purple or black in color. If the twisting has occurred slowly, atrophy of the tumor may follow. If circulation is wholly arrested, gangrene will result; this is an unusual accident; the color alone must not be taken as indicative of gangrene. The inflamed peritoneal surface quickly acquires adhesions, and through this route adventitious circulation sufficient to maintain the vital integrity of the cyst-wall is supplied. The torsion may result in complete severance of the tumor from its pedicle. The omentum is usually found embracing the tumor, and with it the chief vascular connections are formed. Large tumors adhering closely to the parietal peritoneum are confusing and difficult of management. If the hemorrhage into the sac is very profuse, rupture from overdistention may occur.

The symptoms characterizing the accident of rotation of the pedicle are comparatively clear. The diagnosis is more frequently made when the presence of the tumor has been previously recognized. It is not unusual for torsion of the pedicle to

disclose an unsuspected cyst. There is undoubtedly some relation between menstruation and this accident; I have frequently observed the association. The onset is marked by the sudden occurrence of an acute paroxysmal, pulling pain, usually assigned to the side involved, though in one of my cases the pain was referred to the epigastric region only. The severity of the pain varies with the suddenness and completeness of the constriction. Its paroxysmal character probably indicates progressive rotation. The temperature in shock is usually low but may be elevated a degree or two. Vomiting, marked pallor, extreme restlessness, rapid pulse, are the other subjective evidences of abdominal shock. There is dysuria and constipation. The occurrence of syncope and acute anemia would indicate hemorrhage of copious amount into the sac. The muscles of the abdomen are usually tense. The tumor enlarges rapidly, becomes more resistant, exceedingly tender, and fixed. The condition is often mistaken for other acute intra-abdominal troubles, intestinal obstruction, and especially extra-uterine pregnancy. A recent experience of the author will bear detailing in this connection. A lady of 72 years, after two days of vomiting and constipation, and attacks of severe paroxysmal pain on the right side, developed a tumor. It was elastic, distinct in outline, exquisitely tender, about the size of a cocoanut. A diagnosis of twisted pedicle was made. The operation revealed an extraperitoneal hematoma, the hemorrhage coming from the rupture of an atheromatous deep epigastric artery.

The treatment of this complication of ovarian cyst is eminently operative. We must not overestimate the danger of gangrene or rupture of the sac. These extreme results are not so likely to occur as was formerly taught. Therefore, in peracute cases it is well, as Bantock long ago advised, to await reaction before resorting to operation.

Rupture of Ovarian Cysts: Spontaneous rupture of the thin septa is a process by which the locules in oöphoritic cysts are fused into chambers of greater capacity. In multilocular glandular cystomata many small secondary cysts form and may rupture into the peritoneal cavity, giving rise to no phenomena whatsoever. Rupture of a cyst becomes a complication when it produces marked symptomatic effects. Thin-walled unilocular cysts,

those usually designated as parovarian, are especially prone to this accident. Papillary and malignant degeneration weakens the walls and favors rupture. Simple overdistention may result in rupture, but usually the accident is precipitated by violence or by some complication, such as torsion of the pedicle or infection. The contents may escape into the general peritoneal cavity, or through adhesions the rent may occur into the stomach or any portion of the alimentary canal, bladder, vagina, or even through the abdominal parietes.

The consequences of rupture are influenced largely by the size of the tumor, extent and location of the breach, and the character of the escaping contents. Small simple cysts with bland contents may rupture without giving offense. If the escaping fluid is septic, general peritoneal infection is quickly excited. In rupture of papillary and malignant cysts there is diffusion and secondary implantation of the growths over the peritoneum. If the seat of rupture involves a large vessel, fatal hemorrhage may ensue; or there may be general oozing from the congested cyst-wall, due to torsion of the pedicle. Dermoid tumors, owing to their thick walls, are not liable to rupture, yet when there is torsion of the pedicle their sudden distention with blood may lead to rupture, the non-absorbable contents exciting peritonitis. Certain thin-walled cysts, parovarian especially, may upon reaching a certain development spontaneously rupture, to refill and rupture again with marked regularity. The case of Meredith, recorded by Sutton, in which rupture occurred thirty-four times during a period of nine years, is the most striking example of this behavior. Wilson has collected quite a series of these intermittent ovarian cysts, and emphasizes the importance of distinguishing them from intermittent hydronephroses.

This accident is usually easily recognized whether it occurs as a result of violence or spontaneously. It is marked by sudden sharp pain in the abdomen, with immediate alteration in the shape of the abdomen, and generally with entire disappearance of the circumscribed tumor. If hemorrhage occurs there is marked shock. Subsequent symptoms depend upon the character of the escaping fluid. If it is innocuous it is rapidly taken up by the peritoneal lymphatics and eliminated by the kidneys as a profuse diuresis. If septic in character, unless promptly treated

surgically, a general peritonitis and death result. With the escape of the fluid there is collapse of the sac, and the tumor may not reform; but the rent is usually closed by adhesions, and gradually the fluid reaccumulates in the sac. When rupture occurs into a hollow viscus it is usually the result of infection with softening and degeneration of the apposed walls. A profuse diarrhea may be the only immediate symptom. This character of rupture usually results in infection of the cyst with suppuration and permanent fistulous communication.

Pregnancy: While pregnancy is entirely possible at any stage in the growth of a unilateral ovarian cyst, it usually occurs in the incipient period of its development, when the neoplasm is intrapelvic. The local congestion that attends pregnancy stimulates the growth of the neoplasm. The relation that the tumor bears to the gravid uterus depends upon the size of the tumor at the beginning of gestation and the rapidity of its subsequent stimulated growth. If the tumor is intrapelvic and pregnancy should occur, the uterus may enlarge more rapidly than the tumor; in such an event the organ will be displaced forward and upward, and the tumor will underlie the gravid uterus. Upon the other hand, should the tumor be of larger size and its growth more rapid than that of the gravid uterus, if its development is intra-abdominal, it will rise out of the pelvis, occupy one side, and displace the uterus to the opposite side, broadening the lower zone of the abdomen in a characteristic way; this relation may be maintained throughout pregnancy and delivery be successfully accomplished; but the effect of the great intra-abdominal pressure will probably precipitate a premature expulsion of the uterine contents, and should gestation reach anything like full term, labor will be complicated by the deflected axis of the birth-canal, malposition of the fetus, inertia uteri, rupture of the cyst or its axial rotation. After confinement subinvolution, infection, and torsion of the pedicle are the dangers. When pregnancy occurs in a uterus retrodisplaced and compressed by a large and overlying abdominal tumor, the developing uterus may be forced into procidentia, but usually abortion occurs early.

Broad-ligament cysts are not as greatly influenced by the occurrence of pregnancy as those of ovarian origin, but by their

position and the displacement that they produce they render delivery impossible.

The presence of a dermoid tumor is peculiarly offensive to the pregnant uterus; it offers less accommodation, its walls rapidly thicken because of the great vascularity, it excites pain and causes abortion. Both before and after delivery there is especially great danger of infection in these tumors.

If pregnancy is far advanced, tumor small and its position high in the abdomen, operation may be deferred until after the puerperium. If the tumor is discovered in the early months of pregnancy operation should be done regardless of its size or position. All tumors that underlie the uterus or that materially displace the organ should be immediately removed. Broad-ligament tumors should be enucleated if discovered in the early months of pregnancy. If detected after the child is viable, operation should be deferred until near full term, when removal of uterus and tumor will be the proper procedure, which is safer and easier than a Cæsarean section with attempted enucleation through an incision in the broad ligament.

A patient bearing an ovarian cystoma may become the subject of certain complicating conditions which do not in themselves necessarily affect the tumor, but are largely the result of its presence and pressure-effects. Of these we may mention albuminuria, ascites, intestinal obstruction, hernia, and fatty degeneration of the heart.

Symptoms.—**Clinical History:** It is estimated that an ovarian cystoma will terminate the life of its bearer within four years, yet it is also well known that they may exist and cause comparatively little discomfort for thirty, forty, or even sixty years. It is further known that no age is exempt; they have been found in the fetus and in extreme old age. These statements necessarily imply a varied clinical course. There are no subjective symptoms diagnostic of ovarian cystoma. This assertion so repeatedly made by authors has a tendency to lead the operative surgeon to disregard symptoms occurring in the course of their evolution, which may not be in themselves diagnostic, yet which are significant inasmuch as they direct our attention to the accidents and dangers that attend their growth. The behavior of any individual case is contingent upon the circumstance of posi-

tion, rapidity of growth, character of tumor, and the occurrence of complications. Every one has observed individual peculiarities. Women of certain temperament appear to possess a tolerance for abdominal growths which in another would give greatest offense. Generally speaking, nulliparous women present a greater variety of symptoms than do those that have been pregnant. Small tumors of slow development, long resident in the true pelvis, are productive oftentimes of the most marked symptoms at first; later, when they have risen out of the pelvis they may pursue a quiet course until distress results from their size. Thin-walled rapidly-growing single cysts rarely cause much general disturbance. Such cysts when pedunculated adapt themselves to their environments. The fibrous base of some multilocular glandular cystomata, by impaction in the pelvis through the weight of the superimposed cyst, may cause the greatest discomfort by its pressure-effects; the yielding cyst adapts itself to the pelvic and abdominal cavities and its symptoms are late in developing. Intraligamentous cysts, simple or papillary, always displace the pelvic viscera and create pelvic pain. Dermoids, because of their thick walls, resisting contents, and small size, are painful growths; and they are especially liable to infection and to torsion of the pedicle. Papillomatous growths upon the free surface of cysts spread by implantation, choke the pelvis, and produce marked peritoneal irritation.

Pain: An uncomplicated ovarian cystoma of the ordinary type is not productive of great suffering. Actual pain is not often complained of. A sense of discomfort, weight and heaviness, abdominal dragging and backache is more descriptive of the patient's feeling. In intrapelvic growths pain may be referred to the external genitals and extremities. Pelvic inflammation may precede for an indefinite time the development of an ovarian cyst; the long history of pain is then probably due to the tubal and ovarian inflammation and not to the cystic changes *per se,* but the coexistence of the two conditions is always attended by great suffering. Pain is a constant feature in that rare form of tumor, tubo-ovarian cyst. The occurrence of attacks of pain, cutting or lancinating in character, usually indicates localized peritonitis. Colicky pain may denote a hindrance to normal intestinal peristalsis, due to accumulation of gas from pressure or

to constriction from adhesions. Purely cystic growths do not obstruct by pressure. Severe paroxysms of colic occur in the history of small tumors when there is torsion of the pedicle. In inflammation and suppuration of ovarian cysts consequent upon infection, there is either local or general abdominal pain and tenderness according to the extent and degree of infection. The tumor by its pressure-effects compresses and displaces organs, so that their functions are rendered difficult and painful. Intrapelvic growths not infrequently cause dysmenorrhea, painful and frequent urination, and difficult defecation. Intraligamentous cysts, especially of the papillomatous sort, may occlude the ureters, causing hydro-ureter and hydronephrosis, marked by lumbar pain. Dermoids, owing to their small size, density, and liability to complications, are usually attended in their growth by more or less constant pain with intercurrent attacks of acute suffering. A sudden attack of pain following exertion or some unusual violence should suggest the possibility of rupture.

Menstrual Disturbances: The effect of cystic disease of the ovaries upon menstruation is by no means constant. A very small amount of ovarian tissue suffices for its functional activity, yet in rapidly-growing bilateral cysts amenorrhea may develop early. Large cysts, involving one ovary, do not cause amenorrhea except through their effects upon the general system. Amenorrhea should always be held as a suspicious indication of the possibility of pregnancy. Intrapelvic growths are usually attended by menorrhagia, often by metrorrhagia, and dysmenorrhea. In these cases the uterus is displaced, often incarcerated, and its endometrium diseased. Cystosarcoma is nearly always accompanied by uterine hemorrhage, while in carcinoma there is more likely to be amenorrhea. Bilateral disease of the ovaries would occasion sterility. Although unilateral growths, especially the intraligamentous cysts, displace and distort the uterus, yet pregnancy, uterine and ectopic, occurs under almost inconceivable physical conditions. The effect of pregnancy seems to stimulate the growth of the neoplasm, and in many instances very rapid growth has followed confinement. The author removed a tumor weighing over fifty pounds three months and a half after confinement, which was not recognized at the time of delivery.

Digestive disturbances are usually the result of pressure and peritoneal adhesions. Tumors impacted in the pelvis may reflexly cause much digestive disturbance, even nausea and vomiting. The accidents of twisting of the pedicle and infection are always attended by obstinate constipation, nausea and vomiting, and may simulate intestinal obstruction.

Urine: In rapidly growing cysts the secretion of urine is scanty, and usually of high specific gravity. The secretion of a small amount of pale urine of low specific gravity is of serious prognostic import, regardless of the absence of casts or albumin. Albumin in decided quantity does not necessarily affect the course of the disease; it frequently disappears in a short time after the removal of the cyst. Frequent urination may be due either to cystitis or to diminished capacity of the bladder. In a recent case brought to me in which there was a ruptured cyst, tapping had been resorted to every three weeks for nine years, removing not less than four gallons at each tapping; this patient habitually passed less than a pint of urine in twenty-four hours. Preëxisting renal disease has decided bearing upon the operative prognosis. In 32 out of 40 necropsies upon patients who had died after ovariotomy or with large ovarian tumors, Doran found that the kidneys presented very distinct morbid appearances. When ovarian cysts rupture into the peritoneal cavity the fluid is eliminated chiefly by the kidneys, and profuse diuresis is, therefore, one of the leading symptoms of this accident.

Diabetes: Beyea calls especial attention to the relationship of diabetes to certain diseases of the internal genitalia, particularly pseudomucinous ovarian cysts. Tuffier collected 54 cases of diabetes mellitus associated with pelvic tumors; he regarded it as a mere coincidence. Beyea, after a more careful study, also concludes that diabetes is often an independent symptom; but when there is a large amount of sugar in the urine without the symptoms of diabetes, the sugar seems to be dependent upon some specific and separate lesion or abnormal secretion. In such patients the sugar disappears after operation.

Pressure-Effects: It is in cases of large tumors that have reached to the epigastrium, pushing up the diaphragm, expanding the lower ribs, causing alate chest, that pressure-effects are produced. Yet small imprisoned intrapelvic tumors, as we have

seen, have also their symptoms due to pressure. Edema of the lower extremities from pressure on the vena cava, icterus through interference with the portal circulation, and dyspnea from overdistention of the abdomen and displacement of the thoracic viscera, harassing cough, and pulmonary edema or pleurisy, irregular and feeble pulse, and occasionally bronzing of the skin, are some of the distressing effects due to the weight and pressure of the tumor. And if to these we add further the disordered digestion, rapid emaciation, especially noted in the upper thorax, arms and neck, the mental anxiety, the probably hectic condition with slight fever, we should naturally expect to find in one so afflicted that countenance indicative of the mental and physical burden, which Spencer Wells was pleased to consider as peculiar to ovarian cystic disease, and by him designated '*facies ovariana*.'

Physical Signs.—The recognition of an ovarian cystoma rests upon the physical signs elicited. All ovarian tumors at some time in their history occupy the pelvis, yet often so inoffensively that attention is not directed to them until they become of sufficient size to invade the abdomen. The tumor may lie wholly within the pelvis or it may occupy the pelvis and ascend to a variable height within the abdomen, and exceptionally the tumor will lie entirely above the brim of the pelvis.

1. **Small Cysts (Intrapelvic):** Disorders of menstruation, interference with the functions of the bladder and rectum, and a feeling of pressure, backache, and sometimes acute pain are suggestive symptoms that call for physical examination. To detect an ovarian neoplasm occupying the pelvis solely we must rely upon combined abdominal, vaginal, and rectal palpation. It is scarcely necessary here to emphasize the importance of the proper preparation of the patient. It is often wise to supplement the first examination, however satisfactory its revelations may be, by a second examination systematically conducted with the patient under general anesthesia.

The normal ovary cannot be felt, but in its place will be discovered a tumor lying to one side or behind the uterus, attached to the uterus by a tense cord, the utero-ovarian ligament, which is more readily felt by rectum than by vagina. The size, mobility, independent of the uterus, elasticity, smoothness, globular or

ovoid shape, are all determined by combined abdominal and vaginal manipulation. The tumor may lie laterally, but generally behind the uterus in the cul-de-sac. The position of the uterus depends upon the size of the tumor. When sufficiently large, yet still intrapelvic, it displaces the uterus at first to the opposite side and forward; the pedicle of the tumor then lies anteriorly. If the growth is intraperitoneal and the tumor pedunculated, as it increases in size it usually rises out of the pelvis to the front of the uterus, retrodisplacing that organ and forcing it to the floor of the pelvis. So constantly do dermoids behave in this way that Küstner has emphasized it as one of their characteristics. When the tumor is lateral to the uterus a deep sulcus usually demarcates them, and unless adhesions are present the uterus may be drawn down with forceps without influencing the tumor.

These characteristics of an ordinary intrapelvic cystoma are quite distinctive, and are ordinarily easily determined. Obese patients present difficulties in their examinaton. By vaginal examination small papillary cysts and superficial papillomata may be recognized by their bilateral occurrence, insensitive nodulation, and fixity of the uterus from the masses choking the pelvis. When a small cyst becomes imprisoned in the pelvis it becomes tense and hard, the uterus is fixed, the tumor's wall thickened, and it is often mistaken for a fibroid. When the development of the cyst is accompanied by inflammation its walls are thickened, often irregular from adherent viscera, fluctuation is obscured, and the tumor is fixed and tender. Such cases often show symptoms much in common with inflammatory conditions of the uterine appendages. Small dermoids are especially likely to mislead us, owing to their thick walls, slow growth, and susceptibility to infection.

Small intraligamentous cysts are difficult of diagnosis. They do not possess the mobility of an ordinary ovarian cystoma. Occasionally the ovary may be detected behind or to the outer side of the cyst. The uterus is more fixed in a displaced position. The tumor lies low and encroaches upon the pelvic floor, impinging upon the vagina.

2. **Large Cysts**: When the tumor has attained sufficient size to rise out of the pelvis and distend the abdomen its character-

istics are determined by the usual methods of physical examination.

Inspection: Small tumors, the size of a child's head, may occupy a lateral position; but the tendency of a growing ovarian tumor is to assume a central position until it rises above the umbilicus, and then, as Kelly has pointed out, it assumes an inclination to the right. The appearance of the abdomen, distended by an ovarian tumor sufficiently large to reach to the umbilicus or above, is in itself oftentimes peculiar. It rises abruptly from the pubes and slopes gently to its upper limit. This feature is sometimes so marked as to give the abdomen a conical contour; in other instances it is more globular. The highest point is between the umbilicus and pubis, the distance between these landmarks being increased according to the size of the tumor. Tumors of large size greatly distend the abdomen in all directions, expand the lower costæ, and pendulously overhang the thighs, while the intestines occupy the flanks. Cysts of smaller size, of the dimensions of an adult's head, do not efface the waist-line. Broad-ligament cysts do not produce the acute upright distention of the abdomen noticeable in ordinary ovarian cysts because of their peritoneal restrictions and flaccid character. The umbilicus may be pouting. The skin, especially in women who have not borne children, is white, against which background the blue and frequently distended veins stand out clearly. Lineæ albicantes are often present. Occasionally in multilocular tumors irregularities of the surface may be noticed. The tumor is but slightly influenced by respiration.

Palpation: If the tumor is of moderate size, or if the patient is fleshy, or if the muscles are rigid from a recent attack of peritoneal inflammation, the examination should be conducted under anesthesia. Under palpation the abdomen is usually smooth. Unless fixed by adhesions the parietes glide freely over a tumor with distinctly limited boundaries, ordinarily extending into the true pelvis. The entire tumor, if not exceedingly large, may be freely moved laterally. Tenderness is absent unless there is local peritonitis.

To the feel the tumor presents a uniform resistance; this is especially true in all parovarian or unilocular cysts, and quite generally so in the multilocular glandular cystomata, because of

the tendency of the larger cysts to rest in apposition to the parietes; yet it is not uncommon to be able to determine by palpation the multilocular character of the tumor by the detection of the irregularities and bosses yielding different degrees of resistance. The resistance offered under palpation varies much with the character of the tumor. Multilocular cysts with thick walls are elastic; distinct fluctuation may be obscure. Parovarian cysts convey the impression of thin walls and yield exquisite fluctuation. A dermoid, especially when inflamed, may be quite firm and mistaken for a fibroid. Very tense cysts offer great resistance, and they are frequently mistaken for solid tumors. Intraligamentous cysts are usually more flaccid and do not give the same tense feel of other forms. Fluctuation is especially well-marked in all large cysts. In multilocular tumors general fluctuation may be observed because of the polycystic character and colloid contents, yet areas of fluctuation may be located. When ascites complicates ovarian cystoma a double wave of fluctuation is noticed; one is a shallow, superficial impulse, due to the free fluid which is displaced if firm pressure is made, when the deep impulse of the intracystic fluid is obtained.

When complicated by the accidents which may befall these tumors, such as infection and torsion of the pedicle, the soft, fluctuating tumor becomes firm, hard, and resisting. Occasionally the papillomatous character of a tumor may be determined by palpation, only when, however, these growths are upon the outer wall of the cyst. In thin subjects the adhesion of the cyst-wall to the parietes may be determined, but the presence of adhesions may be inferred more from the clinical history than from any physical sign. A fixed tumor in a patient with a history of repeated attacks of localized peritonitis would indicate that the operator will encounter adhesions. Predictions on this point are frequently in error.

A very interesting and unusual sign in broad-ligament cysts has been recently observed by Gersuny; in two instances he observed pulsation in the tumor both by abdominal and by vaginal palpation.

Vaginal examination in cases in which the tumors occupy chiefly the general abdominal cavity gives but little additional information. The uterus may be drawn upward and laterally

displaced, and the whole tumor-mass may lie entirely out of the true pelvis. Kelly emphasizes the fact that small tumors, when they rise out of the pelvis, by traction upon the pedicle draw the uterus to the side to which the tumor is attached. The position of the tumor in the abdomen often misleads us as to the side of its origin. Occasionally an ovarian cyst may acquire adhesions to the liver or stomach and through these attachments be drawn upward entirely out of the pelvis. Such misplacements can only occur in tumors with very long pedicles. In these cases the palpating hand can be insinuated between the tumor and the brim of the pelvis. The difficulties here presented to diagnosis are very great. The history of the case is of some value, but patients' statements as to where the tumor was first discovered are often misleading. In more than one instance women have positively asserted that they first felt the lump of a large multilocular cystoma high up under the liver. The ovarian origin of high-lying cysts must rest upon the inability to feel the ovary of one side, the detection of the pedicle connecting the tumor with the uterus. A tumor occupying both the pelvis and the abdomen will compress the uterus usually into retrodisplacement, which position is best recognized by rectal examination. In intraligamentous tumors the uterus is often carried far to the opposite side, or raised out of the pelvis in front of the tumor. The cervix lies high up behind the symphysis pubis and may not be reached by the examiner's finger. In a case of broad-ligament cyst I found the uterus in procidentia. When the pelvis is occupied by a tumor the bladder is much compressed, displaced, and distorted, a fact that must be remembered in operation.

Percussion: An ovarian tumor that has risen into the abdomen pushes the intestines above and to the sides. Such a tumor is dull over its convexity, the dulness extending into the pelvis as deeply as the pleximeter can be placed, except in those rather unusual cases of high-lying ovarian tumors with long pedicles. The fact that dulness extends from Poupart's ligament and the symphysis pubis upward over the surface of the tumor is significant of its pelvic origin. The central or slightly dextrolateral position of the tumor is determined by the dull percussion-note. Beyond the limits of the tumor, above and laterally, lie the intestines, giving a tympanitic note. This "tympanic corona" is con-

clusive evidence of encysted fluid, the presence of which has been determined by palpation. Occasionally a line of resonance has been found over some portion of the surface of the tumor, due to a prolapsed and adherent coil of intestine. This is very rare. If percussion is made with deep pressure the resonance is effaced and the dulness of the underlying tumor brought out. Another point of great value is the constancy of the dulness, regardless of the posture of the patient.

Auscultation: The absence of auscultatory signs is confirmatory and is of immense value in differentiation. Occasionally we may hear loud friction-sounds produced by the opposed peritoneal surfaces.

Mensuration: The tumor lies in the lower zone of the abdomen and it is this region that is chiefly distended. The distance from the pubes to the umbilicus is relatively much greater than from the umbilicus to the xiphoid cartilage. The greatest circumference in large tumors is at or just above the umbilicus.

CHARACTERISTICS OF VARIOUS VARIETIES.— **Proliferating Cystadenoma:** These tumors do not affect the general health until they are of large size; their growth is comparatively rapid and painless; they are most common between the ages of thirty and forty years, and are of greater frequency than all other varieties of ovarian neoplasms; they attain to enormous size, and yield typical physical signs upon examination.

Parovarian Cyst: These cysts do not impair the general health, their growth is slow, comparatively painless; the sac is smooth, less resistant; the walls of the cyst appear to be thin under palpation, and they are especially prone to rupture; the wave of fluctuation is transmitted with rapidity in every direction; the abdomen has a more globular, less conical shape than in multilocular tumors.

Papillary Cysts: These are common in middle and advanced life; their growth may be very slow until complicated by surface papillomata; they rarely attain to large size; they usually cause great pain, are generally complicated with ascites, and are prone to malignant changes.

Dermoid Cyst: These tumors are frequent in young subjects; they are usually the size of an orange, rarely larger than a man's head; their growth is slow, attended by pain and menstrual dis-

orders; the tumor is frequently enveloped in adhesions, and if of small size it is firm and resisting; the complications of infection and torsion of the pedicle especially befall this variety of tumor.

Differential Diagnosis.—**Ascites:** A knowledge of cardiac, hepatic, or renal disease or of some progressive peritoneal morbid process often anticipates the occurrence of ascites, and the question of differentiation from ovarian tumor rarely arises. When the abdomen is distended by free fluid it is flat, with the patient in the dorsal position, as compared with the conical enlargement of ovarian cystoma. Unless greatly distended the walls are lax, flanks bulging, fluctuation distinct. Percussion shows resonance upon the upper surface; this resonance is somewhat crescentic in shape, with convexity downward. The area of resonance varies with the posture of the patient; the fluid gravitating, the intestines float upon its surface. In some cases of hyperdistention the mesentery will not permit the intestines to reach the surface; resonance may then be developed by deep percussion about the umbilicus. The flanks are always dull with the patient in the dorsal position, while even in very large ovarian cysts some resonance can be detected in one flank or the other. Small ovarian cysts of the papillomatous sort are frequently complicated by ascites. Their diagnosis is rendered easy by elevation of the patient's hips; the fluid gravitating to the diaphragm leaves the lower zone of the abdomen free to palpation, abdominal and combined, a more trustworthy method than the abdominal ballottement employed by Tait. This posture will facilitate the diagnosis of ruptured cysts, as experience has taught me.

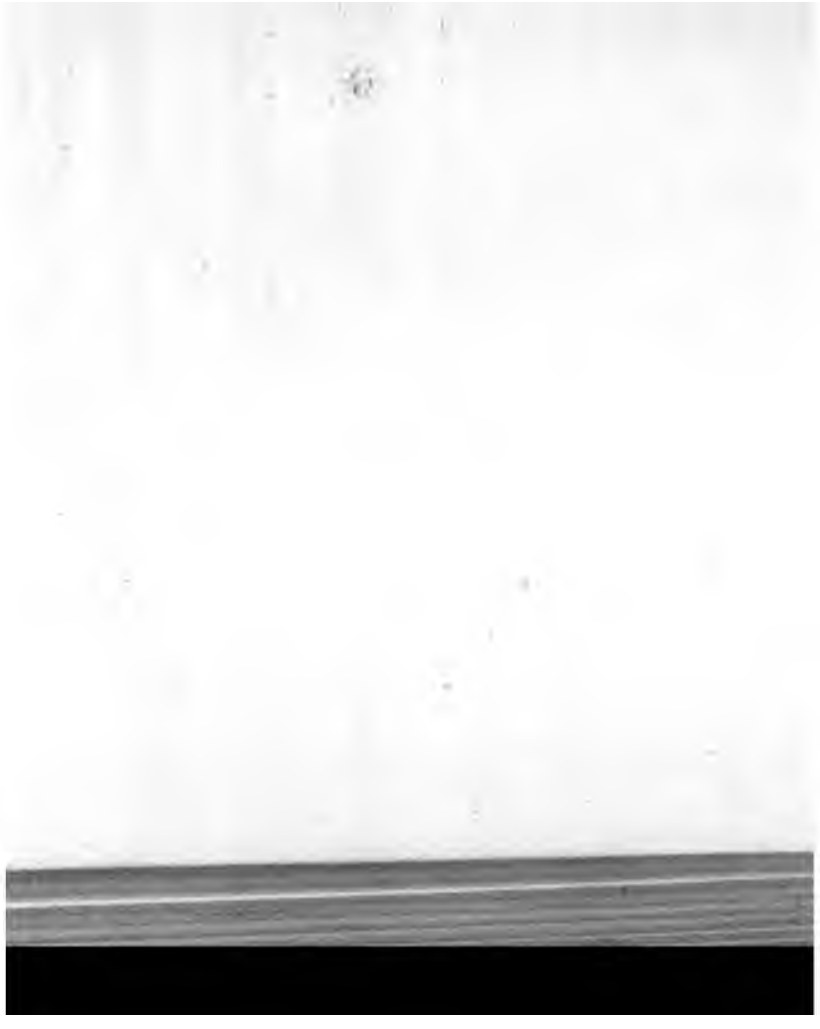
Sactosalpinx: Small intrapelvic tumors may be mistaken for inflammatory conditions of the appendages, especially sactosalpinx. The history of infection, the preceding endometrial disease, the impairment of the general health, the extremely painful, inflammatory, and often acute course of the affection, the intercurrent pyrexia, usually bilateral involvement, fixation of the uterus, shape of the tumor, its connection with the uterus, and sometimes our ability to feel the uninvolved ovary, are our usual points of differentiation. The difficulties of differentiation are

FIG. 1.

FIG. 2.

AREAS OF DULNESS PRODUCED BY ASCITES AND OVARIAN CYST.

FIG. 1, Ascites. FIG. 2, Ovarian Cyst.



sometimes insurmountable when we encounter an imprisoned intrapelvic cyst or small, inflamed dermoid.

Retroflexed Gravid Uterus: The soft, elastic, even fluctuant feel of a retroflexed gravid uterus may be mistaken for an intrapelvic cyst. To guide us aright we have the collateral symptoms of pregnancy, the absence of the uterus from its normal position, the continuity of the tumor with the portio vaginalis, and the possible palpation of both ovaries, and perhaps ballottement. Hegar's sign is not obtainable in this condition.

Uterine Fibroma: Ovarian cystoma may be mistaken for pedunculated subserous fibroma. The growth of a fibroid is usually much slower, and unless imprisoned is not productive of discomfort. The tumor is hard, inelastic, more movable than an ovarian tumor, is intimately connected with the uterus; and frequently the tumors are multiple. The abdomen is asymmetrically distended, and an irregular outline of the tumors may appear. That rare condition, fibrocystic disease of the uterus, may lead us astray. Here the existence of a tumor for a long time, followed by a period of rapid growth, the impaired condition of the general health, its uterine connection, its obscure sense of fluctuation through deep resisting walls, and its nodular surface determine its character. Soft, edematous fibromyomata yield an elastic, even fluctuant sensation under palpation. So deceptive is this that the author has frequently seen the trocar plunged into these tumors after celiotomy. In uterine tumors, especially those of rapid growth, an aortic impulse may be felt, and the uterine arteries are enlarged. Their central position, smooth surface, elastic, edematous feel and absence of fluctuation, great depth of the uterine cavity, frequent metrorrhagia, are the points in differentiation.

Pregnancy: A large ovarian tumor may be mistaken for normal pregnancy. The collateral symptoms of pregnancy may all be wanting, its possibility most emphatically denied, and the fetal heart may not be heard; yet the shape of the tumor which is rather broad above, narrowing into the pelvis, the alternate contraction and relaxation—Braxton Hicks's sign—fetal movements, ballottement, short cervix and soft lower segment of the uterus, are signs which should apparently render impossible the

error; however, but few surgeons of large experience have escaped it.

Hydramnios: This morbid condition which accompanies pregnancy may develop a tumor resembling in many respects an ovarian one. It usually occurs in young women. The general symptoms of pregnancy are usually present; the physical signs of ballottement and uterine contractions are present. The uterine nature of the tumor is established by bimanual examination. Albuminuria is constantly associated with it.

Extra-uterine pregnancy, before the death of the fetus, is determined by the history of the case, palpation of the fetal parts, by the placental souffle, fetal heart-sounds and movements. After the death of the fetus, the history of the case and palpation are usually sufficient to disclose the character of the tumor. Axial rotation of small ovarian tumors may closely resemble, both in symptoms and physical signs, rupture of ectopic gestation. Of course, the knowledge of the previous existence of a tumor would count for much. The quick recovery from shock, and the enlargement of the tumor definitely circumscribed, is a different picture from that which attends rupture in ectopic pregnancy with its recurring syncope, absence of definite tumor if the hemorrhage is intraperitoneal, or the broad, fixed swelling bulging into the vagina, if the hemorrhage is intraligamentous.

Serous peritonitis of tubercular origin often becomes encysted through the agglutination of intestines and omentum. If this effusion is restricted to the pelvis the difficulties of diagnosis are very great, indeed, insurmountable in some instances; but the constancy with which the tubes and ovaries are involved gives the tumor a nodular and irregular feel, differing from the ordinary cystoma. Of great value in these cases are the clinical history and general condition of the patient. In encysted dropsy of the peritoneum above the pelvis we encounter a condition that closely simulates ovarian cyst. Fluctuation may be distinct, and flatness on percussion as marked as in ovarian cyst, yet the boundaries of the tumor are not so definitely limited, fading away into resonant areas, irregular in outline and varying much in examinations at different times, according to the amount of meteorism. Encysted peritoneal effusions are often resonant over some part of their surface and frequently so in all their

circumference; and on deep percussion resonance from the underlying intestines may be elicited through the dulness. Especially valuable in differentiation is the detection of a resonant area between the tumor and the pelvis. It is further important to note the frequent occurrence of these effusions upon the left side; and it is not unusual to find more than one area of circumscribed effusions, producing tumors entirely separate from each other. Additional information is found if the peritoneal disease is associated with active pulmonary or pleural tuberculosis, or with enlargement of the mesenteric glands. The hectic fever that sometimes accompanies this condition is absent in uncomplicated ovarian cyst.

Pseudocyesis and **phantom tumors** are only mistaken by careless and superficial examiners.

Mesenteric cysts usually lie out of the pelvis, the uterus and appendages are demonstrably free, the tumor is often quite movable, usually surrounded by a zone of resonance, and always resonant upon one side.

Hydronephrosis: Investigation will determine, as a rule, a cause for the accumulation of urine in the kidney and the sacculation of that organ. It is necessarily the result of some chronically-acting obstruction to the urinary outflow. This history alone should direct us to the possibility of the renal nature of the tumor. The site of origin of the tumor, its frequently intermitting character, its tendency to retain its reniform outline, the constancy with which the colon is found over the inner or lower anterior surface, and finally cystoscopic examination, are means of differentiation that make error unpardonable.

Pancreatic cysts, developing in the umbilical or left hypochondriac regions, attended by digestive disturbances, and lying under the stomach, displacing the colon downward, inward or outward, can only very rarely simulate ovarian cysts.

Distended Gall-Bladder: Hydrops vesicæ felleæ may attain to such dimensions as to fill the abdomen and true pelvis. These large tumors offer the greatest difficulty in differentiation from ovarian cyst. The gall-bladder may enlarge unattended by the symptoms of pain, jaundice, or colorless stools; the tumor may be first detected at the brim of the pelvis; adhesions may limit its res-

piratory movements; an area of resonance may separate it from the liver. In such cases differentiation must be made by bimanual examination of the uterus and its appendages and discovery of their non-implication. In other cases in which the continuity of the gall-bladder with the liver can be established, when it moves in the arc of a circle with its fixed point at the liver, and in which no pelvic attachment can be demonstrated, the diagnosis is easy. We must not rely in these cases too greatly upon the absence of the usual symptoms of gall-stone; it is in the instances in which these are wanting that the error is made.

Prognosis.—The tendency of all ovarian tumors is to increase in size and by their pressure-effects so to disturb the functions of the abdominal viscera as ultimately to destroy life through exhaustion or superinduced complications. Their average duration is about four years. At any time during their existence life may be terminated by the accidents which may befall the tumor itself or are indirectly the result of the tumor's presence. Malignant changes, especially in papillary cysts, are to be apprehended.

Operatively treated, the prognosis is influenced not only by the skill of the operator, but in some measure by the character of the cyst. An ovariotomy for an uncomplicated, pedunculated ovarian cyst is one of the simplest procedures in abdominal surgery. When complicated by adhesions it may tax those of greatest skill. Intraligamentous cysts are always difficult of removal; the technic now employed, however, has greatly lessened the mortality in these cases. An operative mortality of from 4 per cent. to 10 per cent. for ovarian tumors in general can be claimed. In 394 ovariotomies, collected by Burger, the mortality was 9.5 per cent. The usual causes of death are shock, infection, intestinal obstruction, cardiac failure from fatty degeneration, pneumonia, secondary hemorrhage. Kelly has collected 153 cases of removal of ovarian tumors in patients over seventy years of age, and his conclusion is that age does not materially increase the operative risk. Bullitt, in investigating the influence of the size of the tumor upon the operative mortality, collects 23 cases in which the tumor weighed over one hundred pounds; 21 of these were operated upon; he concludes that the fatality is in direct proportion to the size of the tumor.

Surgical Indications.—The indicated treatment in cysts of the

ovary and broad ligament is extirpation of the neoplasm. There are no contraindications to operation other than those that obtain against any surgical procedure. As indicated in discussion of the complication of pregnancy, delay may be judicious, but the general rule should apply: *ovarian tumors should be removed when diagnosed,* due time being allowed for preparation of the patient. The tumor is exposed by celiotomy. Small dermoid tumors and infected cysts should be removed without aspiration; a large clean incision is preferable to a short one to become contaminated. Large cysts may be forced into the wound, and incised or aspirated and drawn out as evacuated. The peritoneal cavity should be protected by gauze or pads against the cyst's contents. When collapsed the opening in the cyst should be closed by strong pressure-forceps. All adhesions are now separated, the pedicle ligated and neatly covered with its peritoneum, the condition of the uterus and remaining appendages inspected and treated if indicated, and the abdominal cavity cleansed and closed. In papillary cystoma, should the growths have penetrated the cyst-wall, involving uterus, broad ligament and appendages, and in superficial papilloma involving these parts, the condition may be inoperable; but if the mass can be removed, extirpation of the uterus and appendages is the required procedure. Hemorrhage is thus avoided and thorough work is done.

In broad-ligament cysts the aim should be to control the sources of blood-supply before attempting the enucleation of the sac. First, ligate the ovarian artery at the brim of the pelvis on the tumor-side, then the ovarian artery on the opposite side. Now free the bladder and ligate the uterine artery of the sound side. Cut across the cervix and secure the uterine artery on the tumor-side. As the broad-ligament is now open, the cyst may be evacuated and enucleated without hemorrhage. The ureters must be kept constantly in mind. The technic of Dr. Hall, of Cincinnati, is perfect.

II. Benign Solid Neoplasms.

Fibroma and **Fibromyoma**: The existence of pure *myoma* of the ovary cannot be denied, since in the vessels of the organ and on its surface at the attachment of the ovarian ligament muscle-cells sufficient for their formation are found. The purest speci-

mens have been described by Martin and Orthmann, Doran, and Henrotin and Herzog.

Though the myomatous tumors are rare, *fibromata* are more frequently met with; yet Graefe, in the literature of the twelve years prior to 1895, was able to collect only 38 described specimens. Kelly has seen 4 cases. The author has met with only one, which weighed four pounds. Hunner, quoted by Osler, found 10 solid ovarian tumors in the records of 9,400 gynecological clinics at the Johns Hopkins Hospital.

Fibroma of the ovary is most common in the third decade, though not confined to it; Graefe met with it in a subject 72 years of age. Jacobs attributes to a general fibroid change of the genital organs the occurrence of fibroma of the ovary. He has met with 6 cases; in 5 fibroma of the ovary was the sole lesion, and in one there was also multiple fibroma of the uterus. And Weir records a case of ovarian fibroid associated with fibroma of the uterus. True fibroma is a diffuse growth, not encapsulated except by the proper endothelial covering of the ovary. Its development may be very slow, or, in young subjects, quite rapid. The tumor retains its ovarian form in a great measure, is lobulated, usually densely hard, but when of large size it may undergo cystic degeneration or become calcified, altering its consistency. Williams reports 3 cases of calcification of ovarian fibroids, claiming that there are only 8 cases in the literature of the last hundred and fifty years. The fibromata are usually pedunculated; the pedicle may be long, the weight of the tumor, its freedom from attachments, and extreme mobility serving to lengthen the pedicle.

Menstrual disorders are commonly observed in young subjects. In my patient the most distressing symptom was the consciousness of the extreme mobility of the tumor, and an evident tendency to torsion of the pedicle. Ascites is a common complication in this as well as in other solid tumors of the ovary. The diagnosis is difficult, and must rest upon our ability to differentiate it from pedunculated fibroid of the uterus, and from sarcoma. The unimpaired general health and sometimes slow growth are the only distinguishing features from a malignant neoplasm. The association of ascites with ovarian fibroids and its absence in pedunculated subserous uterine fibromata Osler

very properly emphasizes as a differential point of much value.

Fibromyoma, arising from the broad ligament or from the ovarian ligament, is occasionally met with. We must be careful to distinguish intraligamentous fibroids arising from the appendages from those of uterine origin. DaCosta reports 2 cases of fibroma of the broad ligament. Cullingworth's case of fibromyoma weighed seven pounds and five ounces; and Doran's case of fibroma of the ovarian ligament weighed sixteen pounds. In these cases, though intraligamentous, the tumors were pedunculated. Delegrange reports 6 instances of pedunculated fibroma of the broad ligament.

Intraligamentous fibroids are of rapid growth, often contain much muscular tissue, are less dense than fibroids of the ovary, give rise to severe pain and pressure-symptoms, and cause uterine and vesical displacement.

III. Malignant Neoplasms.

1. **Carcinoma:** Repeated references have been made in the course of the article upon "Benign Cystic Neoplasms of the Ovary" to the malignant degeneration of preëxisting tumors, and we have shown that carcinomatous changes are especially liable to occur in papillary cysts; indeed, it is but a slight gradation in these glandular tumors to the atypical carcinoma; the tendency to metastasis in both is further evidence of kinship. Cystadenomata and dermoids, tumors of epithelial origin and characterized by rapid proliferation, may at any time take on atypical formation. While such changes in preëxisting tumors are primary, they develop in neoplastic tissue. Carcinoma of the ovary in its true primary form appears in the ovary not the seat of previous neoplasm. Secondary carcinoma, incident to uterine, mammary and other cancer, is comparatively rare.

Ovarian carcinoma appears in three forms: in the first there is a diffuse cancerous change involving the whole parenchyma of the ovary, resulting in the formation of a solid tumor more or less rounded or oval, sometimes lobulated or nodular, but in a great measure retaining the ovarian shape. The tumor is firm, of small size, rarely attaining to a growth larger than one's fist or a cocoanut, usually lies free in the abdominal cavity, attached only by a short pedicle. Occasionally the disease is bilateral.

Degenerative changes are common, resulting in softening or the formation of cyst-like cavities. Hemorrhages not infrequently occur beneath the fibrous capsule of the tumor, giving it a mottled appearance. Microscopically the growth is medullary, scirrhous, or colloid in character.

The second form evidently originates in the germinal epithelium and appears as a papillomatous excrescence, involving usually both ovaries, and implanting itself upon the adjacent and remote portions of the peritoneum. Metastases in this variety are developed early and are widely distributed.

The third form is the distinctly glandular cancer, the adenocarcinoma, the most malignant of all ovarian diseases. This group consists of tumors which bear a striking resemblance in their external appearance to serous cysts (Rothrock). When the papillary masses predominate and there is but little cystic formation, they are spoken of as adenoid cancer, a solid tumor; but with the formation of the cyst, as is usual, they are called cystadenocarcinomata. According to Pfannenstiel, papillary growths are observed on the external surface of the cyst in half the cases, and, like the papillary cysts, the tumor is of small size with short pedicle and frequently bilateral; but unlike the innocent papilloma, it is rapid in development. These cysts are usually single, but may be multiple. Carcinoma appears at two periods in life, in young subjects and in those of advanced age. Histologically it is an adenocarcinoma, the same tumor presenting a variety of structure. Everywhere there is atypical proliferation of epithelium. The contents of the cyst may be clear fluid, colloid material, or a turbid, grumous material from admixture of blood and degenerated epithelium.

When adenocarcinoma develops either as a primary disease of the ovary or as a degenerative change in a neoplasm, the growth is rapid, metastases occur, cachexia becomes pronounced, and the case pursues a rapid course.

2. **Sarcoma:** Sarcoma of the ovary, while a rare disease, according to Bland Sutton is a more frequent one than carcinoma. Kelly regards it among the rarest of all forms of ovarian tumor. Pfannenstiel found it in 5.38 per cent. of his ovariotomies. It occurs usually as a solid tumor, generally as the spindle-celled variety. When the entire ovary is involved, there may be com-

plete obliteration of the Graafian vesicles, but these may persist and change into large cysts. The cystic character of sarcoma, while generally due to softening and fatty degeneration consequent upon thrombosis and hemorrhage, may also be due to changed follicles. Sarcoma of the ovary occurs at all ages; Doran has described a tumor of this class in a seven-months' fetus, and Kelly has met with it in a patient over seventy years of age. They are often bilateral, especially when of the round-celled variety. A striking peculiarity of sarcoma of the ovary is its frequent simultaneous occurrence in both ovaries; Sutton says that this happens in 20 per cent. of cases. They are usually of small size but may, when undergoing cystic changes, become enormous tumors weighing twenty or thirty pounds. While the pedicle is usually short and adhesions are common, McBurney removed a sarcoma of the ovary with a pedicle six inches long and perfectly free from adhesions; it was mistaken for a tumor of the kidney. Grossly they often appear as a mammoth ovary, and the spindle-celled variety particularly very closely resembles a fibroma of that organ. They are pinkish-white in color, surface smooth, consistency varies with the histological structure; the round-celled variety is soft and resembles brain-structure. Their growth is rapid and metastasis occurs by the veins.

3. **Endothelioma**: As an unusual occurrence there develops in the ovary a tumor grossly resembling fibroma, but microscopically presenting alveoli and cell-structure somewhat confusing to the pathologist. In view of their origin they were designated by Leopold endotheliomata. They are now classed as tumors distinguishable from both carcinoma and sarcoma, yet many differ and consider them as mere varieties of one or the other. Eckhardt holds that they bear a remarkable resemblance in structure to sarcomata; he distinguishes two varieties, the vascular and the lymphatic.

This growth appears as a solid pedunculated tumor, hard or soft in consistency, rarely of large size; its surface is smooth or lobulated; it usually affects only one ovary. Cystic formations are common in the lymphatic variety. A thin capsule invests the tumor, the tumor-tissue proper showing an alveolar or tubular arrangement, sometimes a mere diffusion of endothelial or epithelioid cells in more or less dense connective-tissue stroma.

The cells seem to tend toward a change of their form to the spindle type. In the network of cells old or recent blood-effusions may be found. It is questionable if these tumors deserve an individuality; they should probably be classed as angiosarcomata.

Clinically they behave as malignant tumors, grow somewhat rapidly, produce peritoneal irritation and ascites, are occasionally attended by metastases, and usually recur when removed, though not so certainly as other malignant tumors, from which they cannot be differentiated clinically.

Symptoms.—We may apprehend malignant changes in cystic disease of the ovary when the growth of the tumor, which has been stationary or very gradual for a long period, suddenly becomes rapid, when pain becomes a marked feature, ascites accumulating rapidly with swelling of the extremities; and especially significant is the cachexia or marked anemia. The early history of primary malignant disease is by no means characteristic. In very young subjects tumors of the ovary are usually sarcomatous in nature and bilateral in development. In patients between twenty and thirty years of age carcinoma is more common, and it again appears most frequently in those of advanced age. The growth of sarcoma is favored by pregnancy. Sarcoma may be slow in its development, one to three years; carcinoma is more rapid. Sarcoma forms a solid tumor, usually of flat surface, produces anemia and peritoneal irritation. Sarcoma may attain to large size. Menstrual symptoms are variable; uterine hemorrhage frequently appears as an early symptom, but with the increasing anemia amenorrhea is the rule. In carcinoma there is an irregular tumor, of mixed cystic and solid growth, ascites and edema of the extremities occur early, secondary nodules throughout the peritoneum may be felt, and the development of cachexia is at first slow, then rapid. The occurrence of metastases is common to both varieties of tumor, and indicates a hopeless extension of the disease. Freund lays considerable stress upon the presence of hydrothorax as a symptom of malignant disease of the ovary; he observed it in 10 out of 17 cases, in only one of which was it metastatic; it occurs in both carcinoma and sarcoma. Pritchard, also, has met with it in three cases.

Leopold places the frequency of malignant disease of the ovary

at about 18 per cent. In operations in 131 neoplasms of the ovary Werder found that 15 per cent. of them were malignant. The existence of sarcoma does not necessarily imply a recurrence of the disease after operation. A very interesting case of late recurrence of sarcoma of the ovary is recorded by Miller; the tumor was first removed in 1892; it recurred and was removed again in 1894, to recur quickly; then for 18 months the tumor steadily decreased in size, and the patient's health improved; then there was sudden development of a cough, probably from a metastatic growth, and she died in 1898. Estor has collected 372 reported cases of total or partial extirpation of malignant neoplasms; there were 265 recoveries and 107 deaths, a very high operative mortality; yet it is well known that operations for malignant growths, especially when they invade the ligamentum latum, are very difficult and bloody. 176 of Estor's cases were available for remote results; 104 had recurrence, 59 per cent., the recurrence taking place in less than three months in fully one-half the cases; 72 when last heard from were free from disease, but only 28 of these were under observation for as long as three years. His statistics led Estor to conclude that sarcoma was more favorable for operation than carcinoma.

The diagnosis of malignant disease of the ovary prior to exploration is very difficult. In the absence of metastases, pronounced anemia or cachexia, celiotomy is indicated. The justifiability of further procedure must be determined from the conditions encountered.

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ECTOPIC GESTATION.

DEFINITION.—The fertilization and implantation of an ovum elsewhere than in the uterine cavity constitutes ectopic or extra-uterine pregnancy.

FREQUENCY.—Bandl asserted that extra-uterine pregnancy occurred once in every twelve thousand cases of gestation. In eight thousand pregnancies Price found that eighty-three were ectopic. Such statistics are wholly valueless in conveying a correct idea of the frequency of this affection. It must be admitted that occasional cases of this form of abnormal gestation occur, are not recognized, and are spontaneously recovered from after the death of the embryo. A review of the literature of the last twenty years reveals numberless operations undertaken for this emergency, many distinguished operators being able to record their experience with a hundred or more cases. My work in the last fifteen years has approximated four cases annually.

ETIOLOGY.—That an ovum may become fertilized in any part of the genital tract from the Graafian follicle to the uterus seems to be an established fact. We are not warranted in assuming that normal conception takes place only in the cavity of the uterus, the foundation of Mr. Tait's theory as to the etiology; nor is it plausible to accept the dictum that fertilization habitually occurs about the abdominal ostium of the Fallopian tube. Hofmeier and others have demonstrated that the wave of the cilia in both uterus and Fallopian tubes is downward and outward. There is nothing, then, in the idea that the cilia guard the Fallopian tubes against the entrance of spermatozoa. It is clearly proved that a spermatozoön can swim against the ciliary current and can override the waves of tubal peristalsis. When the site of normal conception is fully determined, perhaps we shall have more definite knowledge upon the question of misplaced pregnancy. We know now, contrary to former teaching, that impregnation may occur in the absence of menstruation. We further know that menstruation usually accompanies ovulation. But Tait, Arthur Johnson, and Martin have taught us that menstruation occurs in-

dependently of ovulation and is an act controlled by a special nerve-center situated in the lumbar part of the cord. There is much dispute concerning the changes that take place in the uterine and tubal mucosa during menstruation. It has been generally held that menstruation prepares the endometrium for the reception of the oösperm; and Tait maintained that a desquamative salpingitis in a like manner prepared the Fallopian tubes. If it is conceded that the menstrual preparation is necessary for uterine pregnancy, we should then consider as a serious etiological factor in ectopic gestation the changes that occur in the tube in those exceptional cases in which it participates in the menstrual act. Henrotin ascribes much importance to this as a cause of tubal implantation. That this menstrual preparation is not a prerequisite for the occurrence of conception all practical workers know. It appears that there is sufficient ground to believe that the fertilized ovum may become engrafted upon the Fallopian mucosa under the same conditions that control its implantation upon the endometrium; in other words, the fertilized ovum can develop upon the normal mucosa of the Fallopian tube, provided it is detained sufficiently long in its transit from the Graafian follicle to the uterus.

The question, then, that concerns us is not so much where fertilization takes place, but why does ectopic gestation occur at all? That the oösperm may develop, Webster claims that it must find lodgment upon tissue capable of giving decidual reaction. Waiving, for the time being, the discussion of the varieties of ectopic gestation, Webster seems to have made out a very plain case. He has demonstrated satisfactorily and conclusively to himself by microscopical study of serial sections of specimens of tubal pregnancy that, as the result of fertilization of the ovum in the tube, there is incited a decidual formation. In normal uterine pregnancy the decidual changes are limited to the uterine mucosa. If fertilization of the ovum habitually or, as now believed, frequently occurs at the fimbriated extremity of the tube, tubal decidual formation would make ectopic pregnancy inevitable in every such case; but this genetic influence, according to Webster, only occurs when there is "some developmental fault whereby there is reversion, either of structure or reactionary tendency in the tubal mucosa, to an earlier type in mammalian

evolution. . . . Because of these occasional changes in the Fallopian tube, conditions are brought about capable of establishing with the fertilized ovum that relationship which is essential to its development." This embryological defect may be considered, then, in the light of a predisposing cause, which only becomes active when from other agencies the fertilized ovum is unduly detained. Sippel very strongly controverts Webster's views, and says that it can now be positively stated that in tubal pregnancy the ovum does not produce decidual reaction in the tube, but an epithelial reaction, as is shown by Peters. Taylor asserts that no one has been able to detect in the pregnant tube anything macroscopically approaching the decidual membrane of the uterus, but that there are local swellings which may mechanically arrest the progress of the ovum in its passage through the Fallopian tube. The hypothesis of Webster explains how the arrested oösperm secures vital attachments and continues to develop, but it is not quite clear to the writer that it is a cause of the oösperm's arrest.

Until something more definite is known, we must in a great measure attribute to mechanical causes the occurrence of ectopic gestation. External transmigration, so strongly pressed by Sippel, recognized seven times out of twenty-five cases by Küstner, regarded as a cause by Martin, and Prochownik, and demonstrated by Williams in five cases out of thirty operated upon by Howard Kelly, appears to be the only possible explanation for those cases of tubal pregnancy occurring upon one side, the ovary of that side being absent, or cystic, showing no Graafian follicle (Bandler), or else showing no corpus luteum, while the unimpregnated tube is sealed at its abdominal end and the ovary of that side shows a recent corpus luteum. In cases of this kind the arrest of the ovum is not dependent upon changes affecting the tube, but the increasing size of the oösperm. In 1856, Virchow attributed ectopic gestation to perimetric adhesions. Since that time macroscopical changes leading to mechanical obstruction are the usually assigned causes. Freund observed that in certain women the development of the tubes is permanently arrested in the infantile state, in which the tubes show a spiral, corkscrew winding, especially toward the abdominal end. These twists cause narrowing of the lumen of the tubes without

disturbing the relations of surrounding parts; and he regards this as the chief etiological factor in extra-uterine pregnancy. Abel, also, considers that the infantile tube is the predominating cause. Other abnormalities of the tubes, such as diverticula and tortuosities, are occasional assigned causes. Herzog says that in a respectable percentage of cases congenital anomalies of the tube are responsible for the occurrence of tubal pregnancy. More obvious causes are polypi occluding the lumen of the tubes, or myomata distorting and compressing them. A condition analogous to congenital anomalies is frequently seen; the tube is in an atrophic state from hyperinvolution, and this form is especially worthy of recognition, says Mr. Taylor, as it has a bearing upon the phenomenon of early rupture.

Mr. Tait, in holding that spermatozoa entered the Fallopian tubes only under pathological conditions, was doubtless in error; and in attributing ectopic gestation merely to a destruction of the ciliated epithelia of the tubes, he but imperfecly ascribed to Fallopian inflammation the importance it deserves in the etiology. With gonorrheal and puerperal infection of the uterus there is extension by continuity to the Fallopian tubes. There is not only endo- but interstitial and perisalpingitis. The lumen of the tube becomes obstructed, its epithelia destroyed, its walls thickened and rigid; peritoneal adhesions contract, distort, and displace the tubes; and subsequently there may be atrophy. In this pathological state we find a complete destruction of those forces, the ciliary wave and peristaltic propulsion, by which the oösperm is conducted to the uterus; and we further have mechanical obstruction to its onward passage.

That tubal pregnancy may occur in an apparently normal tube, one must admit. Martin wholly rejects the inflammatory theory of the causation, regarding the associate pelvic peritonitis as a rapid consequence and not the cause of ectopic pregnancy. A vital blow to the etiological effects of salpingitis is that many pathological reports declare the absence of inflammation altogether. Taylor says: "In 43 cases no certain evidence could be elicited of any preëxisting inflammation." And Bland Sutton writes: "In many instances I have failed even after the most careful microscopical examination to find any evidence of old salpingitis or loss of epithelium." Simple macroscopical exam-

ination is insufficient to determine these changes. No report that is not microscopical, and preferably by serial section, should receive credence.

Orthmann, and Dührssen find in the endosalpingitic alterations an important factor in tubal pregnancies. In 68.7 per cent. of the latter's cases there was preëxisting tubal disease. Stepkowski reports that in 37 cases in which Neugebauer operated endometritis was present in 13 cases, follicular endometritis and ovaritis in no fewer than 11, and pelvic peritonitis in 11; 14 of the 37 women had had abortion in the previous pregnancy; 6 had had pelvic inflammation after previous delivery or abortion. Küstner regards the inflammatory as the most plausible etiological theory. Strassmann views ectopic pregnancy as purely an obstructive condition due to inflammation. And Peterson defends the theory of its inflammatory origin; in 12 of the 14 cases examined there was a mild catarrhal process, which decreased in intensity from the uterus outwards. Schauta believes that in catarrhal swelling of the tubal mucosa, with an open fimbriated extremity, a fecundated ovum in its passage, either directly through the tube or after external transmigration, may be detained in the ampulla because the flow of the stream of serum from the peritoneum into the uterus is broken, and the cilia cannot act against the stasis at the isthmus. Glitsch, who has exhaustively considered this subject, says that inflammatory alteration in the tubes and their neighboring organs is the most prominent cause of tubal pregnancy; and the most frequent microörganism is the gonococcus. The frequent association of salpingitis with tubal pregnancy, the bilateral lesion, the appearance of chronicity of these changes, showing the existence of the inflammation before impregnation, are some of the reasons offered for holding that salpingitis is primary to and probably causal of ectopic gestation.

There is no uniform cause for ectopic gestation, but from the foregoing one must conclude that the chief etiological factors are infection of the genital tract and inflammatory alterations in the pelvic organs.

PATHOLOGY.—The accidents which may befall an extra-uterine pregnancy result in great misplacement of the fetus, placing it at times in almost incomprehensible surroundings. The complex

pathology is greatly simplified if we classify all cases of ectopic gestation according to the site of implantation of the fertilized ovum, and from this pursue it in its various accidental evolutions. Primary abdominal pregnancy is now regarded an impossibility, and is eliminated from the discussion. It must be conceded that ovarian pregnancy is, at least, possible. There is no case on record the authenticity of which is not questioned by some one of high authority; but equally capable and conscientious observers maintain that certain specimens unquestionably demonstrate that ovarian pregnancy does occasionally occur. If not in all, certainly in the vast majority of cases the oösperm finds lodgment in some portion of the Fallopian tube, in an accessory appendage thereto, or in a diverticulum. Primarily, then, there are two varieties of ectopic gestation, *tubal* and *ovarian*.

I. **Tubal Pregnancy:** A fertilized ovum may find attachment in any of the anatomical divisions of the Fallopian tube, and on this basis tubal pregnancy is thus subdivided into *interstitial, isthmic, ampullary,* and *infundibular.* (1) *Interstitial* pregnancy is an uncommon form of tubal gestation. It is often mistaken for cornual pregnancy. The accident of rupture is especially serious in this form and will receive further consideration. (2) *Isthmic* pregnancy, though more frequent than the interstitial variety, is still an unusual form. It occurs in any portion of the tube between the ampulla and the entrance into the uterine wall. (3) *Ampullary* pregnancy is the common variety of tubal gestation. It was, says Martin, the seat of lodgment in 49 out of 56 cases. (4) *Infundibular* pregnancy occurs when the ovum becomes engrafted upon any portion of the pavilion, or, as in Voigt's case, lodged on the fimbria ovarica. Such forms of pregnancy, owing to their adhesions to the ovary, are usually described as *tubo-ovarian.*

When the oösperm is implanted upon any portion of the tubal mucosa its presence excites changes not only at the seat of implantation but somewhat remotely from it. These changes and the accidents incident thereto, which occur in the life-history and after the death of the fetus, constitute the pathology of ectopic gestation.

Changes in the Uterus: The entire genital tract responds to the influence of tubal pregnancy. In a few instances the vagina

appears softer, more capacious, and its mucous membrane of darker hue, but it is especially in the uterus that we note a more significant genetic influence. The entire organ enlarges, its walls thicken, and there is actual hypertrophy of structure and increase of vascularity; its cavity lengthens and broadens. This growth continues so long as the embryo is alive; after its death or removal uterine involution rapidly follows. The uterus does not enlarge to that same degree as in normal uterine gestation. Its cavity rarely attains a depth greater than four or five inches. After rupture of the tube and escape of the fetus into the broad ligament or abdominal cavity, should pregnancy continue, the subsequent growth of the uterus is not so rapid as in the first weeks. All authorities are practically uniform in stating that the uterus invariably enlarges. Granting this to be true, my experience in many cases, in which the duration of pregnancy was three weeks to five months, teaches me that the uterus is not so greatly enlarged as one might be led to infer from the general teaching. The os tincæ is usually patulous, especially if the examination is made prior to the passage of the decidual membrane. In all varieties of extra-uterine pregnancy, says Parry, a decidua forms in the uterine cavity as in normal gestation. This observation of twenty-five years ago, though repeatedly assailed, is an undeniable fact. The decidua possesses the same characteristics as that of normal uterine pregnancy. Its thickness is from 4 to 8 mm., and the formation partly or entirely lines the cavity of the uterus. With interruption of pregnancy there is usually discharge of the uterine decidua. But as this membrane is unessential to the continuance of a tubal pregnancy, it early begins to atrophy and undergo degeneration, and may be discharged during the progress of an ectopic gestation. After the twelfth week the uterine mucosa is usually clear of decidua. The membrane is, as a rule, passed piece-meal; or it may, after liquefying, be discharged as a serosanguinolent fluid. The hemorrhage attending its discharge may be very profuse, and occurs suddenly, to as suddenly cease. In typical cases there is exfoliated a complete cast of the uterine cavity, somewhat triangular in shape and showing the openings for the Fallopian tubes and os internum. The presence of certain large oval cells was considered by Wyder as distinctive of pregnancy. Ayers con-

siders decidual cells pathognomonic of pregnancy, and thinks that the diagnosis of extra-uterine gestation may be made by the microscopical examination of the decidua removed by curettage. Klein denies that decidual cells are pathognomonic. Abel says that the only real difference between menstrual and ectopic-gestation deciduæ lies in the presence or absence of glands.

Webster's observation of decidual formation in the unimpregnated tube is unique.

Changes in the Ovary: The ovary on the affected side in the early stages of tubal pregnancy is enlarged and shows a corpus luteum. In advanced cases it may be compressed and atrophied. In the infundibular form it is frequently attached to the gestation-sac.

Changes in the Tube: It is assumed by Herzog and by Peters that the outer layer of the fetal epiblast, the so-called trophoblast, has a phagocytic or absorptive power which destroys the epithelium and permits implantation of the chorion upon the connective tissue of the tubal mucosa. Stroganowa points out from her own observation that at the site of attachment there is no such development of decidua as in uterine pregnancy. In tubal pregnancy the chorionic villi not only develop up to, but absolutely penetrate the muscular tissue. Though denied by Taylor, Sutton, and others, Webster stoutly maintains that a tubal decidua vera is formed, not necessarily extensive, "more commonly a small circular band around the lumen." The existence of a decidua serotina is now conceded, and placentation probably takes place in the same manner in tubal gestation as in uterine. Webster, Henrotin, and Herzog maintain that a decidua reflexa is essential; but few other observers support this view. Kreisch, however, has observed the formation of decidua reflexa within the tube. Opportunities to observe uninjured specimens in the early stage, before the degeneration of the reflexa, may account for the opposing views. Under the stimulus of pregnancy and the growth of the embryo the mucous membrane is stretched, its plicæ effaced, and the epithelia near the embryo's attachment flattened or destroyed. The connective-tissue cells are converted into decidual cells, and there is increased vascularity and general thickening of the tube from edematous infiltration. The muscular fibers at first are hypertrophied; subsequently the bundles are

broken up, and are gradually displaced in various directions, to finally disappear; so that after a few weeks the gestation-sac consists largely of connective tissue, which may be thickened from inflammation or hemorrhage. The peritoneum is stretched by the enlargement of the tube. There are frequently evidences of peritonitis in the form of adhesions, folds, and twists. While these changes are taking place in the body of the tube, there is great turgescence of the fimbriated extremity, and usually by the end of the eighth week there is retraction of the fimbriæ and contraction of the peritoneal ring, and closure of the abdominal ostium by adhesive inflammation following. In ampullary pregnancies with implantation near the abdominal ostium, this opening remains patulous. The now-known frequency of tubal abortion discredits the constancy of closure of the abdominal ostium. However, it is unusual for tubal abortion to occur after the eighth week. These early changes that occur in the tube are limited to the site of the ovum. In ampullary pregnancies the isthmic portion of the tube may appear normal or may be distinctly atrophied. With continued growth of the fruit-sac there is great thinning of the tube, especially opposite the site of attachment. The intratubular tumor now occupies the same relation to the broad ligament as a hydrosalpinx. In its early stage the tumor is small, appears prominent upon the tube, and very closely resembles, as Mr. Taylor remarks, a subperitoneal myoma. In this stage there is greater vascularity not only of the tube but of surrounding tissues. We have remarked upon the softening and relaxed state of the uterus. To some extent the broad ligaments and mesometric tissues participate in these changes. For these reasons and because of the increased weight of the tube, the tendency is for the tube to prolapse and rest close to the side of the uterus or in the posterior cul-de-sac. By its weight and traction it carries with it the uterus into retrodisplacement. The subsequent position of the uterus depends upon the course of the gestation.

Tubal Mole: If we concede that the fertilized ovum is safely ensconced upon a decidua serotina and provided with a reflexa, its development would proceed, as *in utero,* to maturity if its environments were commodious and the expansion commensurate with the fetal growth permitted. The tendency,

then, of all ectopic gestations is to early interruption; and perhaps the earliest accident that may befall an isthmic or ampullary pregnancy is the formation of a tubal mole. To Mr. Sutton we are largely indebted for the explanation of this termination of tubal pregnancy, which results in the death of the embryo. A tubal mole is identical with a uterine mole. When in the tube it appears as a firm fleshy blood-clot, ovoidal or elliptical in shape, from 2 to 6 cm. in diameter. Within its substance, usually placed eccentrically (Sutton), an amniotic cavity is found, in which we can occasionally demonstrate an embryo. The outer surface, on washing, may show the shaggy chorion. If in doubtful cases, in which there is no amnion or embryo, sections microscopically examined show chorionic villi, usually in clusters, the evidence of pregnancy is conclusive.

The conversion of the pregnancy into a tubal mole is the direct result of intratubal hemorrhage. The source of this hemorrhage and site of collection is a question in dispute. Herzog says: "I think that the extensive hemorrhages from the decidua serotina are of the greatest importance in bringing about the death of the embryo and terminating the progress of tubal pregnancy. . . . These hemorrhages may be very appropriately designated as intervillous hemorrhages." The maternal origin of the blood is established by its character, showing non-nucleated red blood-corpuscles and leucocytes. Mr. Sutton maintains that the initial hemorrhage occurs between the chorion and amnion, in the subchorionic chamber. The blood in such case is fetal in character, showing nucleated red blood-corpuscles and no leucocytes. It is probable that in most instances both observations are correct. The bleeding occurs because of degenerative changes in the villi and from serotinal rents due to stretching of the tube. Whatever the source of primary bleeding, the formation of a tubal mole is often a gradual process, and may become symptomatically recognizable. The blood-clot may appear distinctly laminated. Ultimately there is partial or complete detachment of the delicate chorionic villi, and the circulation of the ovum is thus destroyed and pregnancy arrested.

When apoplexy befalls the fruit-sac, one of several events may take place. In some instances it is quite probable that in the very early stage it may be discharged into the uterus. Under

PLATE XX.

Uterus and Adnexa.

Ruptured Ectopic Gestation.

The fetus occupies the dilated ruptured tube. The interior of the uterus contains the partly detached decidua and the peritoneal surface shows the attached shreds of fibrin due to the plastic peritonitis.

like conditions, in which the hemorrhage is small, and especially if it occurs only in the subchorionic chamber, it is possible for absorption to occur. Usually serotinal hemorrhage, that is, blood from the mother, engulfs the fetal sac and distends the tube, temporarily forming a hematosalpinx. If this occurs at any time prior to the eighth week the abdominal ostium may be open, and through it the blood may slowly dribble into the peritoneal cavity without definite symptoms of hemorrhage; or else, with freer bleeding occurring intermittently, we have recurring shock with each hemorrhage. In this way we have gradually formed a laminated intraperitoneal hematocele. The other consequences of the formation of a tubal mole are tubal rupture and intraperitoneal tubal abortion.

Tubal Abortion: In ampullary pregnancies the formation of a tubal mole distends the tube, sometimes uniformly, in other instances most irregularly. If the distention is symmetrical, which will occur when there is uniform resistance in the entire circumferential wall, we have resulting one of perhaps the most common changes that take place in ectopic gestation; the entire embryo with its membranes is discharged into the peritoneal cavity through the abdominal ostium. Tubal, like uterine abortion, may be a sudden event, or may occur gradually from separation of the membranes by accumulating tubal hemorrhage. The closure of the pavilion usually occurs by the eighth week. That this is not an invariable behavior is shown by the fact that abortions do occur after such time.

The mechanism of expulsion lies in the force exerted by the accumulating blood-clot, which, being uniformly compressed on all sides, is propelled in the direction of least resistance, that is, toward the infundibulum. The muscular wall of the tube, rarefied and succulent from edema, has but little or no power of peristalsis. It is possible that in very early tubal abortion, a few instances of which are reported wherein the gestation-sac, complete or ruptured, is discharged into the celom without hemorrhage (Orthmann), the tube may have some reverse peristaltic power and may vomit its contents. It is more probable that the absence of blood is due to its absorption, and that the abortion occurred in the usual way, the tube being comparatively passive.

I have several times met with complete tubal abortion, twice only with incomplete. In both these instances the mole lay in the grasp of the abdominal ostium of the tube, there was large hematocele, and the tube-wall showed rupture at the seat of primary attachment. In early pregnancy, if the abortion is complete, it is quite probable that the tube, cleared of its contents, will contract, hemorrhage cease, and if the intraperitoneal hemorrhage has been small in amount, absorption occur. Cullingworth, Routh, and others emphasize the fact that the site of rupture of a tube often disappears by reason of the quick involution which follows. I cannot speak authoritatively—I only know certainly that which I have seen. After so-called complete tubal abortions, occurring from the fourth to the seventh or eighth week, in which the embryo in its amniotic sac is found in a mass of blood-clots in the peritoneal cavity, the tube is still enlarged, the infundibulum has a rosetted appearance and is often still bleeding. It is true that in such cases some membranes may be left.

The fetus in tubal abortion has usually perished while resident in the tube, drowned in its own blood or in that of the mother, as you may choose to believe until the point is settled. With its escape into the celom its manner of disposal depends upon the stage of pregnancy, the amount of immediate hemorrhage, and the cessation thereof. An intrapelvic hematocele is formed; if small, it may be absorbed along with the embryo and its membranes; infection may occur, with suppuration; or the blood may be absorbed and the fetus take on some one of the preservative changes. The hemorrhage that attends a tubal abortion may be copious and produce the most alarming symptoms, indistinguishable from rupture; but as preparatory to the actual occurrence of abortion we have happening the intratubal hemorrhages and the formation of a mole, with some bleeding from the tube before the actual delivery of its contents. The symptoms that characterize the slight and recurring hemorrhages should often be correctly interpreted.

Rupture: The isthmic and ampullary portions of the tube are incapable of accommodating the fetus and its placenta beyond the twelfth week. A few exceptional cases may justify the extension of this time to the sixteenth week; Werth has collected

13 cases in which pregnancy had existed six months or longer without rupture of the tube. But at any time after the inception of pregnancy to the third month we may expect the great catastrophe of ectopic gestation, rupture of the tube. This rupture is usually spoken of as the *primary* rupture to distinguish it from *secondary* rupture, which refers to giving-way of the broad ligament after a primary extraperitoneal rupture. Primary rupture in the free portion of the tube, then, may be of two kinds, *intraperitoneal* or *extraperitoneal.*

Primary intraperitoneal rupture in the majority of cases occurs between the fifth and eighth weeks of gestation. The essential cause is the distention and thinning of the tube-wall. Doléris says that the wall is always thinnest at the site of the placental attachment. In early rupture the breach usually occurs here, but in later ruptures it may occur at any portion of the distended tube. The precipitating causes are numerous, and generally involve some muscular effort, such as straining at stool, strained extension of the arms above the head, alighting from carriages and railway trains, etc. Coe especially calls attention to the greater liability to primary rupture at the expected time of the menstrual flow. Through the rent there escapes, partially or entirely, the embryo with its membranes. In some instances a previously-formed tubal mole is extruded. In the later ruptures the fetus often escapes, enveloped in its amnion; or this sac may be destroyed, and the fetus is tethered by the umbilical cord to the placenta, which remains in the tube only partially separated. The rent may be so large on the uterine side of the gestation-sac as to sever the duct entirely.

With the occurrence of the rent there is hemorrhage from the enlarged vessels. If the tear extends into the placental site this hemorrhage is necessarily profuse. Webster asserts: " The earlier in the pregnancy the rupture occurs, the less will be the immediate danger to the mother." This idea pervades all textbooks. Mr. Taylor, in his monograph, is the only author who controverts it, and with his view my experience is entirely in accord. Perhaps one of the quickest deaths after rupture fell to my lot to be witnessed. A nullipara, aet. 21 years, perfectly well and disclaiming any probability of pregnancy, was brought to my office in a collapsed condition, having fainted

a few moments before while shopping in an adjacent store. Dr. Owen Wilson, then a senior medical student, made the diagnosis of intraperitoneal hemorrhage from ruptured ectopic gestation. When I reached her she was in hopeless collapse and died an hour later, two hours from the onset of symptoms. The circumstances were so peculiar that an autopsy was ordered by the coroner. An ampullary pregnancy of three weeks' duration was found. There was an immense quantity of intraperitoneal hemorrhage. In a second case I recognized intraperitoneal rupture and urged operation, which was refused. In six hours from the onset of symptoms the patient was dead. From the examination of the specimen and from all obtainable facts I infer that the pregnancy could not have been of more than four weeks' duration. These autopsies and several operative experiences convince me that early rupture of tubal fetation is an exceptionally dangerous accident, "a special phenomenon of extrauterine pregnancy. . . . As a disease or accident it stands quite alone" (Taylor).

The constancy with which the uninvolved tube is found atrophied or undeveloped explains the thinning and early rupture. These early ruptures, in my observation, have invariably involved the serotinal area. When pregnancy occurs in a diverticulum or accessory tube, early rupture is shown to be the rule. When rupture occurs late, that is, after the seventh week, copious and immediately fatal hemorrhage may occur, but in these late ruptures the placenta may not be involved, or, if involved, may plug the rent. In a similar manner the fetus may be engaged in the opening and temporarily tampon against hemorrhage. It must not be inferred that, in emphasizing the gravity of early rupture, I intend in any way to detract from the serious importance of those occurring later. Parry's statistics reveal the truth of this, and show us that intraperitoneal rupture is generally fatal from hemorrhage within 48 hours. Should the patient live longer than this, recurring hemorrhages, any one of which may prove fatal, are the usual causes of death without the intercurrence of peritonitis.

That an arrest of hemorrhage and interruption of pregnancy with spontaneous recovery has occurred in cases of intraperitoneal rupture is claimed, the fetus being absorbed or else con-

verted into a lithopedion or adipocere-formation. Authors are somewhat obscure upon this point, and such a termination seems very improbable to me. The bleeding is often profuse and so rapid that hematocele is not formed, the blood diffusing generally through the abdominal cavity; or else the hemorrhage is so continuous that the effort at the restriction of blood by adhesions of the overlying intestines and omentum is only partially successful, and we have formed what Mr. Taylor designates an "unstable hematocele." The consistent and usual course after intraperitoneal rupture, unless prompt aid is rendered by the surgeon, is immediate death of the fetus through partial separation of its maternal attachments, and death of the mother from hemorrhage, usually within 48 hours, though life may be somewhat more prolonged, death finally resulting from anemia due to recurring hemorrhages and rarely from suppurative peritonitis.

A combination of circumstances may exist at this time by which both mother and fetus may escape the dangers immediately attendant upon primary rupture, gestation continuing. Tait, and Sutton have both maintained that it is impossible for a fetus, escaping into the abdominal cavity in the early stage of gestation, to retain its vitality. Evidence seems conclusive that this may occur. In order that this may take place the fetus must escape from the tube enveloped in its membranes, possibly only in the amnion, the placenta must retain its original attachment to the tube, and hemorrhage must cease. Though exceptional, this conservative behavior undoubtedly occurs. Taylor collects conclusive evidence, recording cases of his own, of Webster, and even of Mr. Tait. Since his publication, Sutton, in the Transactions of the Obstetrical Society, November 2, 1898, describes a case in which the fetus had escaped from the amnion, and on operation was found alive and disporting itself among the intestines, tethered by the umbilical cord. The demonstration of the amnion in abdominal pregnancy is frequently a very difficult matter, but it is probable that in early rupture the destiny of the fetus rests upon the integrity of the amnion.

Primary Extraperitoneal Rupture: In a certain proportion of ampullary pregnancies which escape intraperitoneal rupture, the growing pregnancy distends the tube and separates the broad ligament, so that the space upon the under surface of the tube,

which in the normal state is a mere line, becomes a wide area. This surface, with obliterated muscle and displaced peritoneum, finally yields to the increasing demands of gestation, and rupture occurs between the folds of the broad ligament into the mesometric space. The subsequent behavior is contingent upon circumstances. The embryo, and the hemorrhage which attends the rupture are now extraperitoneal, confined within a limited space, roofed in by expanded tube and peritoneum, floored by the rectovesical fascia and beneath it the levator ani muscle, and supported laterally by the bony walls of the pelvis. These confines restrict bleeding and favor coagulation of the blood, thus contributing to a spontaneous cure.

The sequels to an extraperitoneal rupture are dependent upon the amount of hemorrhage, the possibility of the continuance of fetal life, the resistance offered by the limiting walls, and the accident of infection. If rupture has taken place with but slight bleeding and complete separation of fruit-sac, the hematoma containing the perished embryo may be absorbed. This favorable course is particularly likely to occur when a tubal mole is discharged. In less favorable cases the hemorrhage is not arrested and bleeding, though intermittent, continues, distending the broad ligament. When, as result of the primary rupture into the broad ligament, we have a sudden large hemorrhage or constantly recurring hemorrhages, the tension overcomes the resistance offered by the peritoneal envelope and rupture occurs through the peritoneum into the general abdominal cavity. The broad ligament may rupture from overdistention at any time in the course of an extraperitoneal gestation. Such a rupture is designated *secondary intraperitoneal rupture,* that is, secondary to primary extraperitoneal rupture, and in contradistinction to primary intraperitoneal rupture. This giving-way of the broad ligament is especially liable to occur as an immediate sequence of primary extraperitoneal rupture. In other instances it ruptures late in the course of the gestation. When it occurs early it permits the discharge into the peritoneal cavity of the blood-clot which has accumulated extraperitoneally, and is attended by fresh hemorrhage, which is equally serious and is symptomatically indistinguishable from primary intraperitoneal rupture.

An extraperitoneal hematocele or broad-ligament hematoma is capable of distending the broad ligament and of elevating the peritoneum from its visceral attachments, encircling and blocking the rectum, and bulging as both a vaginal and an abdominal tumor. A hematocele of these dimensions is laminated, showing recurring hemorrhages. Copious hemorrhage results in almost immediate secondary rupture. When the fetus escapes by primary rupture into the broad ligament, the placenta remaining attached to the tube, the fetus lies amid the connective tissue of the pelvis, surrounded with more or less effusion of blood. If pregnancy is uninterrupted the blood is absorbed and gestation advances as *in utero;* the inevitable consequence is one of two things: either the broad ligament must rupture, or else the pelvic peritoneum must become detached and displaced *pari passu* with the development of the gestation. Secondary rupture is an imminent danger in every case of broad-ligament pregnancy. The gestation-sac, while thickened by an adhesive peritonitis, is very vascular and friable, and in some places especially thin. The placenta in its ectopic site is liable to injury on the slightest traumatism or change of intra-abdominal pressure. From the slightest cause late secondary rupture may occur. Through the rent intraperitoneal hemorrhage usually occurs in sufficient amount to terminate the life of the mother. However, should the rent in the broad ligament take place in a thin area over a presenting pole of the fetus, the fetus may pass through the rent into the general peritoneal cavity; if the amnion is intact its life may be preserved. The conditions then are: the child is in the abdominal cavity surrounded by its membranes, attached by the umbilical cord, which passes through the hole in the broad ligament to the placenta. The continued vitality of the fetus depends upon the condition of the placenta. It must be understood that at all times the placenta must retain in part attachment at its original site of formation. There is no evidence that a placenta can become wholly detached and reimplant itself, yet in all cases of advanced gestation the periphery of the placenta acquires attachments and increased vascularity. Were it not for this appropriating property of the placenta every ectopic fetus would die from insufficient nourishment.

We have so far shown that the immediate effects of a primary extraperitoneal rupture are: first and most favorable, the formation of a hematoma with absorption of embryo and blood-clot. In more advanced cases the absorption of the blood and the preservation of the fetus by conversion into adipocere or into a lithopedion may result. Immediately following the primary rupture, secondary rupture with intraperitoneal hemorrhage and death of the mother is a common termination in this form of pregnancy. Should the ligament resist the strain of the distending hemorrhage resulting from primary rupture, the child being alive, gestation may advance. At any time during the progress of the pregnancy late secondary rupture may result from hemorrhages into the gestation-sac, due to separation of the placenta, or from the overdistention due to the accumulation of amniotic fluid. If this rupture of the broad ligament involves the placenta the mother dies almost invariably from the fearful hemorrhage that occurs. In other instances the thin peritoneum may suddenly or gradually yield, and the child escape into the abdomen, the case proceeding as one of abdominal pregnancy; Michie's case is a typical illustration of this variety, although the child was dead at the time of operation. There yet remains another course of behavior for an extraperitoneal rupture. Under favorable conditions, after rupture into the broad ligament, the fetus may survive and pregnancy advance as a true broad-ligament or tubo-ligamentary pregnancy, that is, the placenta remains attached to the tube and the fetus is extruded into the broad ligament. This condition is sometimes described as subperitoneopelvic, which it is at first, but, as Mr. Taylor says, this term is inadequate if the fetus continues to grow, and it is therefore spoken of as subperitoneo-abdominal; it seems that the term 'broad-ligament pregnancy' is comprehensive and quite sufficient.

In this form of pregnancy the placenta usually overlies the fetus, and gradually, as the gestation advances, the peritoneum is raised from its visceral and parietal attachments to accommodate the advancing gestation. Hart and Carter's, and Webster's sections show the peritoneum raised from the uterus, bladder, and anterior and lateral abdominal wall, so that a large portion of the gestation-sac lies in front of the peritoneum in direct contact with the anterior parietes. These sections demonstrated so

clearly this anterior evolution of broad-ligament pregnancy that it has been assumed by many that this is the usual form of development. Dunning, referring to Hart and Carter's case, published in 1883, says that he has collected 52 cases of advanced broad-ligament pregnancies, and that in only 2 did the fetus and placenta bear such an anatomical relation to the abdominal wall. He then describes his own case which is typical of posterior ligamentary evolution, wherein the peritoneum was raised from the pouch of Douglas, from the rectum, uterus, and posterior pelvic wall as high as the sacral promontory. The releasing of the peritoneum from the sacral promontory, as Mr. Taylor remarks, seems to be sufficient to accommodate the gestation. Taylor confirms Dunning's assertion that posterior ligamentary development is the most common mode of invasion in a broad-ligament pregnancy. The placenta in the early stage of broad-ligament pregnancy, as previously remarked, usually overlies the fetus. In the abdominal evolution, whether by anterior or posterior ligamentary evolution, the fetus ascends and is generally found higher and freer in the abdomen than the placenta. Should a broad-ligament pregnancy from any cause perish prior to maturity without rupture, the fetus and its secundines may remain inoffensive for an indefinite time; but usually the cause leading to the death of the fetus is degenerative changes in the placenta, giving rise to hemorrhage, which, though not necessarily sufficient to cause rupture, is very likely to invite infection, and we have the whole mass undergoing suppurative changes.

Intraperitoneal Hematocele: Reviewing the causes of intraperitoneal hemorrhage, we find that the bleeding may arise in connection with tubal mole, tubal abortion, and tubal rupture. In retained tubal mole the hemorrhage may be very slow and may accumulate in the pelvis. A similar behavior exceptionally attends tubal abortion. With rupture of the tube there is, as a rule, such copious hemorrhage that nature hasn't time to confine it to the pelvis. A pregnant tube usually occupies a retro-uterine position, and rests in the cavity of the cul-de-sac. Mr. Taylor's original drawings very correctly illustrate the conditions usually encountered. But occasionally we find that the tube, intact or ruptured, occupies an interior position, in relation to the bladder and anterior surface of the uterus. Doran, Meek, and McLean

report such cases in the advanced stage of pregnancy. I have seen this relative position only in the early stage.

The position of the tube, preëxisting adhesions, and the rapidity of the blood-effusion determine the position, shape, and stability of intraperitoneal hematocele. If conditions are favorable for blood-coagulation, the tumor lies behind the uterus and generally pushes that organ forward, sometimes raising it entirely out of the pelvis. The uterus may lie centrally, or be quite laterally displaced. If the uterus is found to be central in position the hemorrhage is probably intraperitoneal. In other instances the uterus is covered and forced downward and backward by the accumulating hematocele. In escaping from the tube the blood accumulates in the cul-de-sac, the intestines are floated out, and there is quickly excited an adhesive peritonitis, agglutinating intestines and omentum, fundus uteri, bladder, and anterior parietes, the whole soldered mass covering and encapsulating the slowly accumulating blood. In the center of the blood-clot lies the tube, and from it, either through rent or abdominal ostium, from time to time fresh hemorrhage is poured out, so that the fresh blood accumulates centrally, and the temporary limitations yield for its accommodation. In this way a distinct intraperitoneal tumor may be formed, irregular in outline, elastic or fluctuating to the feel. Inevitably more copious hemorrhage will occur, rupturing the temporary limiting walls and deluging the peritoneal cavity.

The influence of preëxisting adhesions upon the position and direction of burrowing of intraperitoneal hemorrhage has been remarked by all operators of experience. In a case upon which I operated six days after the primary rupture, there was a history of long-standing pelvic peritonitis. The uterus was firmly fixed in retrodisplacement, the omentum was widely attached to fundus uteri, bladder, and anterior abdominal parietes, concealing all of the left tube and ovary; the pregnant right tube was only partially covered by omentum. The rupture had occurred upon its anterior peritoneal surface, and in the rent there lay a fetus two inches long. The effused blood had escaped between the omentum and the anterior parietes, and lay as a shield, two inches thick and firmly coagulated, over omentum and intestines. Three pints of this coagulum were removed

before securing the tube. The omentum had so thoroughly protected the general cavity that there was no blood in the pelvis or among the intestines. This case presented many confusing physical signs, though the history and general symptoms enabled Drs. Buist, Wilson, and myself to quickly agree upon a diagnosis of tubal pregnancy with rupture. The disposition of the blood-clot and displacement of uterus and appendages in this case is, perhaps, unique, yet it illustrates in a most striking manner the influence exerted by previous adhesions upon the disposition of effused intraperitoneal blood.

A gravid tube, prolapsing to the side and behind the uterus, folds over it both layers of the broad ligament, and prior to rupture or leakage adhesions may form between the broad ligament, omentum, viscera, and posterior parietes, thus excluding the ovary, tube, and its contained fruit-sac from the general cavity. Should bleeding occur under these circumstances, encapsulation is probable. It is this state of affairs which is so frequently mistaken for extraperitoneal rupture. I know the error is common. In ruptures, early or late, but especially in early ones, with free bleeding there is no effort to a pelvic restriction, and we find the blood filling the lower spaces of the abdomen; and while generally diffuse, blood, differing from ascitic fluid, shows a tendency to accumulate in certain areas, and owing to its consistency, to gravitate more slowly to dependent positions. It is only, then, in moderate hemorrhage, with nature assisting to encystment and pelvic restriction, that we have a distinct pelvic tumor, which bulges the vagina and is constant on change of posture, formed from an intraperitoneal rupture or abortion.

It must be admitted that intraperitoneal hemorrhage may be occasionally due to reflux menstruation, and is sometimes occasioned by a ruptured hematoma ovarii, the pathological status of which is not understood. Barring these extremely exceptional causes, and, of course, traumata, intraperitoneal hematocele is practically universally due to tubal gestation. It is somewhat surprising, then, to see a recent statement from Webster, that in 146 cases of intraperitoneal hemorrhage collected from various sources, only 28 per cent. were due to ectopic pregnancy. Webster considers these statistics misleading, and they certainly must be, being disputed by Cullingworth who performed abdominal

section in 20 cases for hematocele, and in every instance it was associated with tubal pregnancy.

Broad-Ligament Hematoma: In 83 operations for ectopic gestation Price states that he has never seen an extraperitoneal rupture. When you open the abdomen for supposed tubal pregnancy and find lying to one side the dark-purple tumor, the round ligament passing over its anterior surface, the Fallopian tube somewhat expanded and enlarged upon some portion of its upper surface, the ovary usually flattened and displaced toward the outer pelvic wall, the uterus generally elevated out of the pelvis, displaced to the side opposite to the tumor and occupying the same relations to the tumor as it does to an intraligamentary cyst, and further, when the peritoneum is found raised from the posterior surface of the uterus and from Douglas's cul-de-sac, and in some cases the anterior reflection of the broad ligament elevated from its cellular attachment, and if by incision over the tumor through one layer of peritoneum liquid or clotted blood is exposed, you have in this demonstration, proved by actual findings, sufficient criteria upon which to base the diagnosis of an extraperitoneal hematoma.

It must be granted that inflammation and adhesions may so thicken and alter the capsule that it is sometimes difficult to ascertain these determinate points, but any argument that attempts to sustain the position that tubal pregnancy never ruptures downward between the folds of the broad ligament is a reflection upon the integrity and intelligence of the vast majority of surgeons engaged in this line of work.

The condition is closely simulated by intraperitoneal rupture amid preformed adhesions, above described. A broad-ligament hematoma accumulates subperitoneally between the layers of the *ligamentum latum* laterally and above, and the fascio-muscular pelvic floor below. It dissects around the rectum, raises the peritoneum from the recto-uterine space, elevates the uterus, sometimes burrows beneath its posterior peritoneal envelope, and makes its way into the opposite side. Usually the mass is localized upon one side, flat upon its upper surface, rarely rising above the brim of the pelvis, and bulging the vagina as an elastic tumor. When the effusion occurs upon the left side, through tension upon the pelvic fascia there is annular constriction of the rectum. If

operators would make autopsies upon some of their cases of supravaginal hysterectomies, they would occasionally find opportunity for studying this picture of extraperitoneal hematocele, and would learn the dissecting power of the blood so confined.

It is hardly fair to state, though practical work would indicate it, that intraperitoneal hematocele is always associated with tubal pregnancy. On the other hand, extraperitoneal effusion, that is, broad-ligament hematoma, is a common occurrence, due to many causes, but only occasionally to tubal rupture.

Infection: Extraperitoneal hematocele, from its relation to the rectum, is especially liable to infection; and its conversion into an abscess is a frequent sequel. On the other hand, suppuration is extremely unlikely to occur in intraperitoneal accumulations of blood, because patients usually die from hemorrhage before infection can occur; yet when pyosalpinx exists upon the same side, as is occasionally seen, or, as very frequently happens, involves the unimpregnated tube, we have the chance of infection from this source as well as from contiguous and involved intestine. In those cases in which pregnancy continues after rupture either as a tubo-abdominal or as a broad-ligament pregnancy, the gestation-sac is constantly exposed to the danger of infection, precipitated by slight hemorrhages or bowel-pressure. After the death of the fetus, either before or after full term, the causes leading to death may invite infection. Usually when suppuration involves the fetus the process is a very slow one, about 60 per cent. of the mothers dying from septicemia. The abscess containing disintegrated fetus may open through many avenues, usually into the large intestine or rectum, or else through the abdominal wall or vagina. Tuttle recently describes a case in which the entire fetus was expelled by the rectum. Baatz reports a case in which the tubal sac opened into the bladder and pieces of bone came away during micturition; with the aid of the cystoscope he removed large masses of bone and pus from the bladder. He is able to find 13 similar cases reported. Infection does not necessarily follow the death of the fetus. The placenta may become thrombosed and atrophied; there is gradual absorption of the amniotic fluid, and the fetus itself may undergo mummification, become a lithopedion or adipocere, in which state it may remain quiescent for years. Dr. Fort recently reported to the Nashville Academy

of Medicine the death of a woman through suppuration and exhaustion attending the discharge of fetal parts; this woman I had examined fourteen years before after spurious labor, and had recognized extra-uterine pregnancy.

A retained fetus is a source of constant danger. Atrophy and complete absorption of the placenta should be expected after the death of the fetus. These changes, however, do not invariably occur; indeed, the thickest portion of the placenta is often at the site of its acquired blood-supply, and the placenta has been known to enlarge after fetal death. It is now held that there is no actual growth of placental tissue, but an increase in size may be noted, due to blood-extravasation and edema. Suppuration in the presence of extra-uterine gestation is not necessarily connected with or caused by the gestation. In the etiology we emphasized how frequently the appendages were found diseased, and while acknowledging myself somewhat behind the times, I must hold that inflammation is in many instances responsible for the misplacement. It is, therefore, not surprising that cases are occasionally met with showing advanced inflammatory processes involving the tube. Noble, and others have reported cases of tubal gestation in one tube and pyosalpinx of the other. A very unusual case, but not at all improbable, according to the author's conception, is that reported by Hare, in which there was pus in both tubes with pregnancy in the fimbriated extremity of the right tube. He thinks that infection followed the impregnation in this instance.

There is evidently a tendency to recurrence of ectopic gestation. But few authors have failed to meet with one or more instances; I have met with two instances in fifty-eight cases. Varnier and Sens have studied 96 cases, reported as repeated ectopic gestations. They retain 65 of these as beyond dispute, all bilateral. More unusual, however, are cases of same sided and same sited tubal pregnancy. Stahl reports a very interesting case of this nature, and refers to the reports of Haydon, Coe, and Henricus of similar cases. Coe's case, while same sided, was not same sited. Stahl's patient had three celiotomies within eighteen months, two for right sided tubal pregnancies, and one for left cystic ovary. The occurrence of early tubal abortion certainly explains the possibility or even liability to a reoccurrence of

pregnancy in the same tube. The explanation of the tendency to repeated gestation is probably bilateral atrophy or insufficiency of development. Though numerous cases are reported of repeated pregnancies, in 1899 Ferguson could collect only 14 cases in which laparotomy had been performed twice. Surely his work was not thorough. Pregnancy may be encountered in both tubes. In Noble's case on one side the gestation was supposed to be of about ten weeks' duration, and on the other of about four weeks. And there may be twin pregnancy in the same tube (Henricus). Lately comes the report from Dr. Krusen, published in American Medicine, January 4, 1902, in which the Fallopian tube contained triplets, apparently in the second month of gestation.

Pregnancy may occur concurrently in the tube and uterus. This most unusual condition has received analytical study by Dr. Gustav Zinke, of Ohio; and his paper before the Pan-American Medical Congress, in Havana, 1901, is an exhaustive *résumé* of the subject. From all sources he collects 88 authentic cases, including one of his own. When the pregnant tube ruptures early the uterine pregnancy is not necessarily interfered with. Celiotomy may relieve the emergency, or, as in most instances of this complication, the rupture has been intraligamentary. In only 26 of the collected cases did the uterine pregnancy advance to full term, and in 18 only did both fetuses develop to the end of the ninth month. If rupture occurs early into the peritoneal cavity, the symptoms of intraperitoneal hemorrhage will probably be so impressive as to thrust a true diagnosis upon us, regardless of the possibility of intra-uterine pregnancy. When, however, the symptoms of rupture are slight and its consequences delayed, either by the continuation of the ectopic gestation or by conservative preservation of the ectopic fetus, the advancing uterine pregnancy conceals the complicating condition, and the true status of the case is not recognized until after the uterus is emptied, either prematurely or at term.

Interstitial Pregnancy: If the fertilized ovum becomes attached and develops in that portion of the Fallopian tube traversing the uterine wall, it is designated an interstitial pregnancy. The gestation-sac must lie between the mucous membrane and peritoneum, and should, if properly restricted, show muscular covering on all sides. The tumor is intimately connected with and is

a part of the uterus; the round ligament and Fallopian tube lie to the outer side of the gestation-sac. Many cases, pronounced interstitial, are instances of pregnancy in one horn of a bicornute uterus, or else are isthmic pregnancies that have invaded the uterus. In the latter class Mr. Taylor places Cullingworth's case, and I am quite sure now, after my studies upon this subject, that a case reported by me as interstitial was really isthmic in character. This form of pregnancy is exceedingly rare; there are but very few cases reported. In 1324 cases of extra-uterine fetation Beckmann found only 40 of the interstitial variety. One of the most accurately described and unquestioned cases is recorded by Dunning. Mr. Tait says: "An interstitial pregnancy ruptures uniformly, so far as we know, into the peritoneal cavity," and this rupture always occurs before the fifth month. Mr. Taylor, also, asserts that the only rupture that is known to occur is into the abdominal cavity. Other authorities contend that in advanced gestation rupture may occur into the uterus, or simultaneously into the uterus and peritoneal cavity. There are three cases on record, quoted by Dunning, of rupture into the broad ligament. Intraperitoneal rupture of an interstitial pregnancy behaves in every way similarly to an intraperitoneal rupture of the free portion of the tube. It is, perhaps, more immediately fatal from excessive hemorrhage. Its other features are covered in the general pathology already given.

Ovarian Pregnancy: The question of ovarian pregnancy will not down. The statement that it is possible but very improbable, so repeatedly made in all text-books, conveys but little information. If there is such a pathological accident as the fertilization of the ovum in a Graafian follicle, then it is far more frequent than is imagined, and blood-cysts of the ovary will probably be found to be the consequences of it. Sutton, and Doran for a long time maintained its impossibility, but both concede now that it does occur. From personal examination of the specimens of Van Tussenbröck, and of Anning and Littlewood, Sutton regards them as incontestable proof of the existence of ovarian pregnancy. Professor Galabin has always maintained that ovarian pregnancy did occur. Gilford, in a review of practically all collected cases, especially of the two above referred to, that of Dr. Croft, and one of his own, concludes that there is

such a thing as ovarian pregnancy, and that it is not so rare an occurrence. He collects 16 cases which he regards as instances of true ovarian pregnancy, and 12 that are probably so. Webster, as late as 1901, in reviewing Tussenbröck's case, denies that it in any way establishes the existence of ovarian gestation. And he maintains on phylogenetic grounds that there is strong presumption that the fertilized ovum in the human subject can begin its development only in tissues derived from the Müllerian tract.

SYMPTOMS.—Any woman of procreative age and physiological genital functions after insemination may become the bearer of an ectopic fetus. It is met with in early sexual life and in those approaching the climacteric, but it is most common between the ages of 25 and 35 years. Statistics show that those who indulge in illicit intercourse are somewhat liable to this misfortune. It may be observed in the nullipara and in the multipara. A period of sterility, varying from one to many years, often precedes the occurrence of ectopic gestation. So frequently do we find this recorded in the clinical histories of these cases that it is now regarded as the rule. Due attention and significance should be attached to it, yet we should never esteem it of essential value. We find mention of ectopic gestations occurring in the first months after confinement. In several instances I have met with it close upon the heels of abortion. That pregnancy may occur the general system must approach something near a physiological basis, and, as some one has remarked, prior to the occurrence of ectopic gestation women often declare that they were in excellent health, in better, perhaps, than they had been for months. This in no manner excludes previous inflammation of the genital tract, a condition we have elsewhere admitted as having a distinct etiological bearing in a certain number of cases. This previous state of well-being merely argues an arrest or period of quiescence in the infection. In others the history is clear of anything approaching a genital disorder.

The symptomatology of extra-uterine gestation embraces the symptoms of the pregnant state and the symptoms of its ectopic character. Those symptoms and signs, local and reflex, responsive to gestation, are not diagnostic of the condition under consideration; they are but contributing evidence in establishing the proof of pregnancy. This evidence is often precluded on physio-

logical grounds; or, more clearly stated, a lactating woman with normally suspended menstruation would, through the breast-signs and menstrual functions, furnish no clue to a state of pregnancy. It is still more important that we appreciate that in the early stage of ectopic fetation, that is, within the first three months, the period of greatest danger, many, if not all, of the collateral symptoms of pregnancy may be wanting. Upon the other hand, patient inquiry may elicit symptoms indicative of pregnancy, which will open our minds to the formation of proper conclusions. The symptoms of uterine pregnancy are evolved in its several stages, and these, though somewhat imperfectly, apply to ectopic gestation.

Fifteen years ago there were certain signs and symptoms which were regarded, when taken collectively, as pathognomonic of ectopic gestation. How many cases fail to be recognized, measured by this standard, one is unable to say. The profession was then only beginning to realize the value of Tait's teaching. In latter days there seems to be a well-organized crusade determined to despoil the so-called classical symptoms of ectopic gestation. Every feature has been attacked and shown to be of inconstant value. It would seem that, as the result of this discredit of the value of the symptoms, there would be fewer correct diagnoses; but just the reverse has followed. We now hold each symptom of some value, no single one is considered pathognostic; and the group of symptoms, regarded as classical, is rarely met with in clinical histories. It therefore appears to be appropriate to consider in detail the phenomena encountered in the progress of an ectopic gestation.

Phenomena of Normal Pregnancy: In the early months of ectopic fetation the condition of pregnancy may be emphatically impressed upon the patient, if she has experienced maternity; or other multiparæ disclaim the possibility of pregnancy, declaring that they have not the general sensations usually present. The irregularities of menstruation may, in one instance, strongly point to pregnancy, and in another discredit it entirely. The special disturbances of constipation, vesical irritation, nausea, are variable symptoms, often wholly absent in early ectopic pregnancy, while in some patients they may be quite as characteristic as in uterine fetation. The breast-changes are especially deceptive in

the early months, and to them we can attach but little importance prior to rupture. In uninterrupted gestation, after the viability of the child, the certain signs of pregnancy may be obtained: fetal heart and placental sounds, fetal movements, and palpation of fetal parts. These merely indicate that there is a state of pregnancy with living child; its location must be determined by other means.

Pain: A comprehensive idea of the morbid changes in ectopic gestation will in a great measure explain the causes and character of the pain, which is present at some time in its history. Pain may be due to conditions preëxisting and wholly apart from the pregnancy. The presence of peritonitis, salpingitis of the affected or opposite side, would account quite sufficiently for the pain experienced. That such pain is intensified under the turgescence incident to the pregnancy, without any necessarily marked change in the tube or fruit-sac, seems most reasonable. In retrodisplacement of the uterus, fixed by peritoneal adhesions, pain is experienced and becomes more noticeable on account of the increased vascularity of that organ. I think that in many cases of very early pain we have in peritonitis a sufficient explanation. Pain has been attributed to uterine contractions; undoubtedly this is one element in its production. Free uterine hemorrhage, with discharge of clots and decidua, excites labor-like pains. Tubal contractions are often considered the cause of colicky pains. The tube, no doubt, is at times capable of muscular work, but the effect of a growing ovum in the tube is to destroy its muscular coat, hence other reasons must be found to explain the colicky pains. Janvrin long ago attributed colicky pains to slight hemorrhages, distention of the tube, and slight laceration of nerve filaments. Tait considered them due to tears. These slight pains, often recurring and colicky, are quite characteristic of the formation of tubal mole, with an effort at tubal abortion, and are therefore often felt in the early stages of gestation. Another cause of most pronounced pain is laceration of the peritoneum and the affront to it due to the sudden inpouring of blood into its cavity. Pain is oftentimes due to pressure, especially marked when caused by extraperitoneal hematocele. The pressure upon and displacement of the uterus and bladder causes characteristically heavy, aching pain. The ten-

sion, constantly increasing in those cases in which gestation continues, creates a sense of discomfort, which rather distinguishes it from a normally placed pregnancy. The compression of intestines and ureters causes pain, local and reflex in character. Constantly during the progress of an advanced ectopic gestation pain may occur from slight hemorrhages into the placenta. And, finally, we have the pain of spurious labor, due to uterine contractions, violent fetal movements, and not infrequently to slight hemorrhages. After the death of the fetus great suffering may result from pressure and inflammation.

The seat of the pain is the lower zone of the abdomen, usually felt "all across." It is not, as a rule, distinctly localized to one iliac region. In cases of rupture the pain may be referred to other regions; in one of my patients the pain was felt in the epigastric region only. In advanced gestation there may be pain from pressure, referred to the extremities and genitals. If the ureter is involved the consequent hydronephrosis may cause a lumbar pain.

The character and quality of pain is interesting. From it we may sometimes judge of the nature of the changes taking place. The pain of rupture is usually a sharp, stabbing, lancinating, terrible, agonizing pain. This pain we have above described as being due chiefly to the injury to the peritoneum, and it bears but little relation to the amount of intraperitoneal hemorrhage. The colicky pains, so conspicuously mentioned in all text-books, are especially indicative of the formation of tubal mole and the gradual occurrence of tubal abortion. These colicky pains occur at frequent intervals, many times during the day, and are usually attended by slight depression. Their frequent repetition and transient effects distinguish them from the sudden and violent pain of rupture, which, if recurrent, will be at longer intervals, of days or even of weeks. Prior to rupture and perhaps prior to hemorrhage in the gestation-sac, pain is described as burning and sometimes as throbbing. This is due to the increased vascularity and mild peritonitis excited. The labor-like pains are especially characteristic of tubal abortion in the early stage; and in the last stage of pregnancy they occur with the effort at spurious labor. The accidents of ectopic gestation are usually attended by pain, yet there are cases that reach a dangerous stage

in which pain is insignificant, or, as Boldt has observed, entirely absent. It cannot be categorically stated that the pain of ectopic gestation in any stage is distinctive in character. It has a peculiarity, however, which is of significance, and that is its sudden unanticipated onset.

Shock: Inseparably connected with the symptom pain is the condition of shock. We so often hear or read the statement that in extra-uterine pregnancy "shock means hemorrhage" that we are prone to attribute the vital depression exclusively to the loss of blood. That this is not always true, experience attests. The degree of shock in many cases of rupture is out of all proportion to the amount of blood extravasated. And we also have shock, mild in degree, attending the pains that occur from formation of a tubal mole without tubal rupture. It therefore appears that the injury to the peritoneum, either from complete rupture or extreme distention, as in intratubal hemorrhage, is an element in the production of shock. Yet it must not be denied and it is safe to hold that shock of deep degree indicates tubal rupture or tubal abortion with free hemorrhage, and that progressive and continued shock unmistakably denotes unarrested hemorrhage. In the early stage, prior to rupture, and in those cases in which rupture is very slight, the patient complains of pain, perhaps colicky, sharp or lancinating, with faintness and slight nausea, and she may pass into temporary unconsciousness; from this she reacts. These phenomena may recur at variable intervals, may be through a period of several days or weeks, until the final catastrophe of rupture occurs, with severe hemorrhage, when she passes into profound collapse. With many patients we have not these prodromal warnings. Following a violent and sudden onset of pain, she falls to the floor in a state of syncope, exhibiting all the characteristics of acute anemia. Sudden and profound shock, occurring within the first four weeks of tubal gestation, indicates tubal rupture with severe intraperitoneal hemorrhage. Milder degrees of shock, between the third and eighth weeks, argue the possibility of tubal abortion. After the eighth week shock indicates rupture. Extraperitoneal rupture, with its necessarily limited amount of hemorrhage, is not attended with the same severity of shock as is intraperitoneal rupture. It is exceptional for patients to succumb to the primary shock without

exhibiting some reaction, though of temporary character. I make this statement advisedly, that reaction is to be anticipated. Yet, as elsewhere noted, I have seen two patients die in this initial shock.

The pulse, temperature, behavior, and mental condition of the patient are our chief guides. The pulse becomes small and running in character. The arterial tension is low; it strikes with a little jump and subsides again. If hemorrhage is continuing the shock will be progressive. If you count the pulse every ten minutes you will find its rate increasing. The general appearance of the woman indicates a condition of acute anemia; her conjunctivæ, lips, and gums are almost as pale as the surrounding skin. Her eyes are sunken and anxious in expression, her respiration sighing, with an occasional deep, long-drawn breath, her voice is husky and she speaks in a whisper. Her surface is cold and clammy, her fingernails are colorless, her temperature subnormal. In some cases the patient lies very still and quiet; others are extremely restless. Toward the end, which is indicated by the flickering and wavering pulse and dilated pupils, she becomes delirious.

Patients who react after the primary shock remain in a condition of anemia. Usually this is enhanced by uterine hemorrhage and by slight recurring internal hemorrhages. Often there is decided reactionary temperature; I have several times found copious intraperitoneal hemorrhage followed by a temperature of 102° or 103°. This pyrexia does not necessarily indicate infection or peritonitis, but is due to the absorption of the extravasated blood.

Menstrual Disorders: There is no one symptom connected with ectopic gestation concerning which there have been such contradictory statements as that of the nature of the menstrual disorders. The reasons for this are obvious. There is no constancy in case of this symptom. Weindler observed 50 cases with reference to the character of the disorder present; in 18 there was amenorrhea. In Hirst's list amenorrhea was present in about 70 per cent. In Fränkel's there was amenorrhea in 26 out of 54 cases. In my own experience there has usually been delayed menstruation for a week or two, and after that time irregular uterine hemorrhage. It has rather been the exception

for patients to pass over two periods. In some instances the menstruation has occurred regularly at the expected time. Usually, if the period is delayed, say a week or two, when it does occur the uterine hemorrhage is often quite profuse and more protracted. Following this rather free bleeding there may be spotting noticed for variable intervals, with reoccurrence of free uterine hemorrhage. The blood is generally dark, thickish in its consistence (Cullingworth). It occasionally comes in gushes; patients have been known to die from uterine hemorrhage in tubal gestation. The quantity of blood lost, as a rule, is not great, however, and this is one of the features that distinguishes it from abortion. The occurrence of the uterine hemorrhage, Mr. Taylor thinks, indicates some disturbance in the gestation-sac, and generally with free uterine hemorrhage and discharge of decidua there is termination of the pregnancy. Gestation may progress, however, after the uterus has cast off its decidua. With the onset of violent pain, such as may attend rupture or abortion, there is, as a rule, a discharge of blood from the uterus. I should say, then, that in the majority of cases there is usually some delay of the periods, after which irregular uterine hemorrhage is the rule. Coe's statement that rupture is very likely to occur at the time of the expected menstrual period is well sustained. After primary rupture, should pregnancy continue, amenorrhea becomes the rule. It is not alone upon the quantity, the time of occurrence, and the consistency of the uterine hemorrhage that we rely for aid in the diagnosis of ectopic gestation. The bleeding comes from the uterus and is due to the separation of the decidua. The finding of shreds or casts of this membrane in the uterine discharges is valuable evidence. In many cases the decidua is passed unawares; it often escapes observation; Grandin recently says that he has seen it but once.

We have discussed in the pathology the diagnostic value of the decidual membranes. It is sufficient to say here that as a means of determinate diagnosis the microscopical examination of membranes from the uterus has proved disappointing. But when we can obtain the history of the discharge of such substance or find portions in the blood-clots passed, irrespective of the microscopical showing, it is of value when taken in connection with other signs.

The Uterus: The condition of the uterus and its position depend upon the stage of gestation, the relations of the gestation-tumor, and, if rupture has occurred, upon the direction of hemorrhage, whether extra- or intraperitoneal. In the early weeks the mucous membrane of the vagina and cervix shows but little change in color. After the second month this is quite noticeable. The cervix is usually hard, but if hemorrhage is in progress and the decidua is being cast off, the cervix may be soft and os slightly open. It is generally stated that the uterus is enlarged, but in the first six weeks of gestation it is a very difficult thing to determine this enlargement. If we have reason to suspect that pregnancy has existed six or eight weeks, more significant than the enlargement, is the shape of the uterus; it is not anteflexed with elongated supravaginal cervix and soft, globular enlargement of the body; though enlarged, it retains the shape of a non-pregnant uterus, and I have not found this enlargement to be of such degree that one could definitely declare to it; it is certainly not in keeping with the stage of pregnancy. The cavity of the uterus increases in depth in early pregnancy from 3½ to 4 inches; in advanced pregnancy it rarely measures more than 6 inches. The position of the organ is more significant than its size. Prior to rupture the uterus is movable. It may be and generally is displaced to the side opposite to the pregnant tube. In intraperitoneal hemorrhage it may be forced forward to the pubis and elevated out of the pelvis. If previous adhesions and retrodisplacement exist it may underlie the blood-collection. If the pregnant tube falls forward, the uterus will lie in retrodisplacement. In extraperitoneal hemorrhage the uterus is displaced laterally and is often elevated out of the pelvis.

Tumor: The tumor of ectopic gestation is formed by the misplaced pregnancy as it develops in its primary and secondary positions. There is also a tumor-like formation due to the confinement and coagulation of blood, which has effused in consequence of rupture or intraperitoneal abortion. The embryo and its sac at the fourth week give a tumor about the size of a pigeon's egg; by the sixth week it has become as large as an English walnut; in eight weeks, as large as a hen's egg; in twelve weeks, as large as a man's fist; in four months, as large as two fists (Smolsky). These approximate dimensions will give us some idea of the size of the tumor to be

found before the accident of intratubal hemorrhage. In the early weeks a pregnancy resident in the free portion of the tube develops a distinct, movable, sometimes apparently pedunculated tumor. It is ovoid or globular in shape. Frequently in this stage it lies quite laterally to the uterus or else closely behind it. It is sharply demarcated from the uninvolved part of the tube, and is elastic or boggy in feel. Extremely characteristic and important is a sensation of pulsation imparted to the examiner's finger by the enlarged arteries. Contractions of the tube are said to have been felt. The tumor prior to rupture is usually described as tender or sensitive; Janvrin especially insists upon this quality as a feature present before rupture. Others, Mr. Taylor among them, assert that pain is indicative of some change occurring in the gestation-sac, leading to the formation of mole. My opportunities for deciding this question have been limited; in the only cases observed prior to rupture, the very sensitiveness of the tumor led me to think that it was inflammatory disease of the tube. The mobility of the tumor may be limited by previous adhesions. Its position may be lateral to the uterus, behind it, or, exceptionally, forward in the uterovesical pouch. We frequently find the opposite adnexa normal. It is valuable and guiding information if upon the affected side we can find two lumps, one the ovary, the other the gestation-sac.

With the occurrence of intratubal hemorrhage within and around the fetal membranes the tumor rapidly enlarges, and now the tube becomes distended in a great part of its length; it is tense, hard, irregular, generally sausage-shaped. It is no longer movable, quickly acquires adhesions, and usually lies in the cul-de-sac, there to await the indefinite but certain accident of rupture or abortion. When tubal abortion occurs, either before or at the time of escape, the hemorrhage, if moderate, accumulates in the pelvis. Intraperitoneal hematocele is generally attributable to tubal abortion or to rupture with moderate bleeding. It must not be inferred that tubal abortion gives rise only to moderate hemorrhage; on the contrary, it may be as copious and continuous as that following rupture. If from either of these accidents or from secondary rupture, then, there occur free and repeated hemorrhages, there is but little time for the formation of pelvic hematocele. The torrent of blood deluges the cavity. There

is no distinct tumor-formation, but an ill-defined mass. The abdominal muscles are rigid at first, later relaxed. There is central resonance, with iliac and flank dulness, which dulness slowly disappears on change of posture (Robson). The vaginal vault seems distended and full, but not resisting; upon elevation of the patient's hips the fulness disappears. The uterus is not necessarily displaced. And more to the point, these cases of copious intraperitoneal hemorrhage are often due to early rupture; in such cases after rupture we may fail to find any tumor.

When, however, the hemorrhage is slow, and especially when it is confined by preëxisting adhesions, we then have the formation of a conglomerate tumor, consisting of the fetus and its membranes, the enlarged tube, and coagulated blood. This tumor is situated in the cul-de-sac at first, gradually fills the entire true pelvis, and is covered by adherent omentum and intestines. Its upper border is rather flat and stretches across the lower zone of the abdomen; or it may lie laterally, extending above the brim of the pelvis into the iliac region and forming a visible and palpable tumor. It bulges into the vagina, and forms a convex, boggy, doughy mass posterior to the uterus, usually pushing that organ forward, sometimes upward and laterally. We may sometimes feel the fetus through the vagina. Bimanual examination shows the tumor to be indefinite in outline.

If the rupture has occurred between the layers of the broad ligament we have a distinct tumor-formation. Immediately after the occurrence of the hemorrhage the swelling is soft and elastic; later, it becomes more resisting, harder, and irregular. It bulges into one side of the vagina, displaces the uterus extremely to the opposite side, and often elevates it out of the pelvis. By bimanual palpation the swelling is convex and smooth upon its upper surface, fading off into the iliac region. The whole mass is fixed, immovable, and lies distinctly to one side of the pelvis. It may increase rapidly with recurring hemorrhage, spreading behind or to the opposite side of the uterus; or, with arrest of bleeding, its disappearance from day to day by absorption is noticed. Both in intra- and extraperitoneal hematocele I have noticed discoloration of the surface of the abdomen. Very different from intraperitoneal hematocele, this, the extraperitoneal, causes great pain by pressure and tension upon the overlying peritoneum. It

constricts the rectum about four inches from the anus and renders defecation difficult.

With living fetus and continued broad-ligament pregnancy the gestation-sac at first lies in the pelvis, occupying the same relation to the uterus as does a small intraligamentary cyst; but as pregnancy advances, obeying the usual law of posterior ligamentary evolution, the tumor assumes a central abdominal position, the uterus then lying immediately in front of it or laterally. The tumor now has its individual characteristics: it contains a fetus whose separate parts may be palpated; owing to the thinness of the gestation-sac, they appear to lie immediately beneath the parietes. Ballottement may occasionally be elicited per rectum or vagina. Fetal movements are felt, and heart-sounds are often to be detected. Placental souffle may be heard, but the position of the placenta deep in the pelvis may obscure this sign. In advanced gestation the uterus is enlarged sufficiently to be felt above the pelvic brim, recognized by its shape, position, contractions and relaxations, which phenomena have been observed in the gestation-sac, also. After the death of the fetus, as is recognized by the disappearance of heart-sounds and placental souffle, and by cessation of stirrage, there is absorption of the amniotic fluid and rapid decrease in the size of the tumor.

Muscular Rigidity: This is a sympathetic response, and is noticeably prominent only in those cases of sudden intraperitoneal rupture; it is only transient then if shock is profound. It must not be mistaken for the voluntary contraction, a protective effort of the patient against rude examination.

Tenderness: After rupture the abdomen is immediately hypersensitive, often over its entire surface. In profound collapse all sensibilities are obtunded. The tumor formed by hematocele is usually very tender over its abdominal surface, much more so than by its vaginal approach. Should infection occur, the whole abdomen becomes exquisitely sensitive.

Exploratory Operation: Should symptoms prove indefinite and physical signs obscure and inconclusive, if the patient is in a precarious state, we are fully warranted in resorting to exploratory operation. Opening of the posterior vaginal cul-de-sac will disclose hemorrhage and permit examination of the adnexa. In

cases of doubt this route is preferable to the abdominal for diagnostic purpose.

Clinical Picture before Rupture.—Ectopic pregnancy prior to rupture has been repeatedly recognized and successfully treated surgically. One operator, Dr. Baldwin, of Columbus, Ohio, has reported 11 such cases. It has not fallen to my lot to make this early diagnosis.

When a patient in comparatively good health, with a previous history of pelvic disorder and perhaps sterility, consults you, giving a menstrual history of amenorrhea varying from a few days to two or three weeks, or else of protracted menstruation with continued spotting, and complaining of vague atypical, not necessarily colicky pains in the pelvis, of peculiar sensations, morning nausea, and sore breasts, we should think of the possibility of ectopic gestation. The pelvic examination, showing a slightly enlarged uterus, a small, movable, elastic, pulsating and sensitive tubal tumor, lying laterally to or behind the uterus, the contiguous ovary separately palpable, and the opposite adnexa healthy, we have sufficient evidence to warrant a provisional diagnosis of intact ectopic gestation.

If the pains become more conspicuous and of a more definitely colicky character, if uterine hemorrhage is more profuse and contains decidual membrane, if the tumor is larger than the probable stage of gestation warrants, and if such tumor is oblong, sausage-shaped, extremely sensitive, hard and tense, the inference is that there has been intratubal bleeding, that gestation is at an end, and that a tubal mole has formed.

When a patient with the above history suffers with severe colicky pains, or with pain of an entirely different character, sharp and cutting, attended by slight attacks of syncope, which show marked tendency to recur at varying intervals; and when upon local examination we detect a boggy mass behind the uterus, the presumption is that upon operation we shall find intraperitoneal hematocele, due to partial or complete tubal abortion.

Clinical Picture at the Time of Rupture.—The symptoms of primary intraperitoneal rupture are those of intraperitoneal hemorrhage, characterized by severe stabbing, lancinating, agonizing pains, felt in the lower portion of the abdomen, sometimes quite definitely upon one side, occasionally quite remotely re-

ferred, accompanied by shock, usually somewhat prolonged, rarely deepening into profound collapse; further evidenced by acute anemia, with its rapid flickering pulse, ashy-pale face, blanched mucous membranes, cold extremities and low temperature; by hurried respiration, thirst, perhaps nausea and vomiting, sometimes great restlessness, or else an oblivious apathy, occasionally passing into a wild delirium and ending in death. More commonly there is slow reaction. The patient shows the effects of the storm through which she has passed by her great prostration; she complains of dizziness and "blind spells"; her bowels are constipated; frequently there is retention of urine; she also complains of soreness in her abdomen, which is found to be rigid and hypersensitive, or, if a few hours have elapsed, there is some distention, and we may be able to detect by the slowly changing dulness in the dependent regions the evidences of free fluid. Vaginal examination often shows a soft cervix with slightly gaping os, an enlarged uterus, behind which there is a sensation of fulness, and sometimes a definite mass can be outlined. A distinct tubal tumor may be felt if the gestation has advanced beyond six weeks. But not infrequently the pelvic examination is unsatisfactory, if not entirely negative. It is very probable that, while conducting this examination, fresh hemorrhage will be excited, the patient will complain of faintness, and her pulse will increase in frequency. These symptoms may be elicited immediately at the time of rupture, or several days may have elapsed. Usually their severity receives prompt diagnosis and treatment, or else death removes the patient from our sphere of observation.

Should, however, as occasionally happens, rupture take place into the broad ligament, the pain of onset is not so severe, but that following is more continuous and distressing than in case of intraperitoneal rupture. So, likewise, is the shock and the consequent anemia less pronounced. Abdominal rigidity is not so likely to be present, and subsequent distention is not so noticeable. Sensitiveness is more intense, but rather localized. There is no evidence of free fluid in the cavity. Bimanual examination shows a distinct tumor, at first tense and elastic, subsequently boggy or hard, lying laterally to the enlarged uterus and displacing that organ upward and to the opposite side. From day

to day changes are noticed in the tumor; it either increases from recurring hemorrhage, which is also marked by the general symptoms, or lessens in size; and unless overtaken by the accident of infection, if the fetus is dead, the tumor may disappear through absorption, or remain indefinitely enclosing its preserved contents.

Should the broad ligament become overdistended from recurring hemorrhage or from the growing fetus, secondary rupture may occur into the peritoneal cavity. In all cases in which it follows as the immediate result of the primary rupture, the symptoms of this secondary rupture are identical with and indistinguishable from those of primary intraperitoneal hemorrhage. The diagnosis of the secondary rupture can only be made by a knowledge of the previous state of the patient. When a broad-ligament pregnancy, through continued growth, bursts its peritoneal envelope, the event may be characterized by severe and fatal hemorrhage if the placenta is involved. Should it occur through the thin peritoneum, not involving any large vascular trunks, the fetus may be released from its subperitoneal position, to take up its abode within the abdominal cavity.

Differential Diagnosis.—**Intra-uterine Pregnancy:** Under normal conditions in the early months there should be but little difficulty in distinguishing uterine pregnancy from ectopic. Bimanual examination showing the compressible zone between the hard cervix and globular body, so characteristic of intra-uterine pregnancy about the eighth week, is not simulated by the symmetrical uterine enlargement of ectopic gestation. The soft, fluctuating body of the pregnant uterus is never encountered in extra-uterine fetation. The finding of an extra-uterine tumor merely indicates a complication. The nature of that tumor, whether a gestation-sac, inflammatory, or neoplastic, remains to be determined. In some cases of pregnancy with scanty amniotic fluid and thin uterine walls, the facility with which the fetal parts are felt may create the impression that the sac is ectopic. The history and careful bimanual examination, revealing the uterine character of the tumor, are sufficient to make this diagnosis.

A more common error is to mistake a pregnant laterally-flexed uterus for ectopic gestation. This error has been made by the

most experienced obstetricians. Fabricius has been twice deceived, and advises that, to avoid the error, we should seek to follow out the round ligaments, a sign not always obtainable. Lateral flexion is usually attended by adhesions, and to distinguish it from an ectopic gestation we should rely chiefly upon bimanual examination. The absence of the uterus from its proper position, the intimate connection between the cervix and the lateral tumor formed by the enlarged body, and the fact that there is but one tumor—the uterus itself—should, with the aid of the clinical history, prove sufficiently determinate.

When uterine pregnancy coexists with prolapsed and enlarged ovary or with small cysts of the ovary, the diagnosis is more difficult. In such cases there may be pains which closely simulate those of ectopic gestation, but the changes in the ovary or tumor are not such as characterize the behavior of an ectopic gestation, which is succumbing to the accident of intratubal hemorrhage.

A confusing condition arises when we meet with uterine pregnancy in a retroflexed organ. With incarceration of the uterus in this position we have all the atypical pains that are met with in ectopic gestation. There may be with such uterine gestation slight hemorrhage or spotting. The tumor in Douglas's cul-de-sac, soft and boggy, resembles the hematocele from ectopic gestation. The cervix lies forward, is often hard and elongated, and its upper portion may be mistaken for the body of the uterus, the fundus being considered as extra-uterine. If, under anesthesia, careful bimanual examination is made, if need be in genupectoral position, the diagnosis can be determined. In the event of great doubt abdominal section would be preferable to the use of the sound, as by it we might liberate the incarcerated uterus and avoid abortion, or, if extra-uterine fetation is found, apply the proper treatment. The essential point in differentiating a retroflexed gravid uterus from an ectopic pregnancy in Douglas's cul-de-sac is finding the uterus separate from the tumor in the latter condition.

When appendicitis complicates uterine gestation in the early months the condition very closely simulates tubal pregnancy, especially tubal abortion. The onset of pain in both may be very sudden, and the location and character of the pain closely re-

semble. The general symptoms of pregnancy may be present in both. The pain and vomiting of appendicitis may cause hemorrhage from the pregnant uterus, and this feature adds greatly to the confusion in diagnosis. A pelvic tumor is not often found in appendicitis, but may be occasionally. The chief differential points lie in palpating the normal adnexa, in determining that the gestation is intra-uterine by the physical characteristics of the pregnant uterus at that stage, and in the localizing symptoms, such as point-tenderness, unilateral rigidity, and perhaps a tumor in the appendicular region. Lastly, the general state of the patient must be considered: in perforative appendicitis there is shock, usually with quick reaction and temperature, the primary pallor is but slight, the color is rather dusky and cyanotic, while the pulse may be very slow, or fast during shock, slowing with reaction, possessing volume and strength; and there is no recurrence of fainting or shock. These symptoms are very different from the classical symptoms of tubal rupture or abortion.

Uterine Abortion: The clinical history in uterine abortion may be identical with that that characterizes rupture or tubal abortion in ectopic gestation. The physical signs are altogether different and are usually sufficient for discrimination. We should urge the greatest caution in the use of the uterine curette if there is the slightest possibility of ectopic gestation. Such patients should be examined under anesthesia, and a properly conducted bimanual examination is the safest method of making a diagnosis.

When, however, uterine abortion is complicated by an adnexal tumor the difficulties of diagnosis may be insurmountable. We should rely upon the general state of the patient for indications for interference, and if this warrants delay, repeated examinations at short intervals will discover any changes in the tumor. Uterine hemorrhage and pain sufficient to resemble an abortion never occur in ectopic gestation without some change going on at the site of pregnancy.

Pyosalpinx: In analyzing the symptoms of ectopic gestation, we have noted that all signs of pregnancy may be absent, that uterine hemorrhage, in place of amenorrhea, is common, and we have frequently referred in the pathology to the fact that pelvic inflammation often precedes ectopic gestation. Recognizing

these clinical facts, it is not surprising that tubal disease, especially pyosalpinx, may be mistaken for extra-uterine pregnancy. A sudden invasion of the peritoneum from a pyosalpinx, either by leakage or extension through the lymphatics, may excite the most violent peritoneal pain, which seizure of pain is not infrequently attended by uterine hemorrhage. Knowing, then, the uncertainty of the clinical history, the two conditions, as so far revealed, may be identical. Generally a pyosalpinx that is so active as to simulate ectopic gestation will disclose a history indicative of infection: fever, sweats, and perhaps chills. These symptoms may have existed for some weeks before the acute peritoneal assault; whereas in ectopic gestation the health of the patient immediately preceding the occurrence of the pregnancy is usually good. In both, too, there is a tumor which may be exquisitely sensitive. The physical characteristics of these tumors may very closely resemble, but in pyosalpinx we never find the soft, elastic, pulsating tumor present in extra-uterine pregnancy. Pyosalpinx is generally bilateral, while in tubal pregnancy the opposite tube is frequently normal. The accident to a pyosalpinx, leading to pain and shock, is invariably followed quickly by high temperature, flushing of the face, with a full pulse; but the temperature in ectopic pregnancy is normal or subnormal at the time of rupture, and there is no pyrexia immediately following. The shock which attends a leaking pyosalpinx is not followed by recurring attacks of syncope.

If pyosalpinx complicates tubal pregnancy a difference is to be noticed in the tumors of the opposite sides, and the general symptoms will indicate the accidents of rupture or abortion.

Ovarian Cysts with Torsion of the Pedicle: This accident may befall any form of pedunculated ovarian cyst, but is especially likely to happen to dermoids. Recalling how frequently these tumors in their growth are attended by pelvic pain, we can fully appreciate that, with torsion of the pedicle and hemorrhage into the sac, the picture may closely simulate that of ectopic gestation. I have twice met with this condition in cases in which the question of extra-uterine pregnancy was raised. The menstrual history and the absence of all signs of pregnancy (yet we know that in ovarian growths there is sometimes colostrum in the breasts) are sufficient generally to exclude tubal pregnancy with

rupture. In torsion of the pedicle the pains are distinctly colicky, vomiting may occur, the abdomen is rigid, and, more confusing, there may be recurring syncope. The tumor is not as intimately connected with the uterus as is a gestation-sac, the uterus is not enlarged, the tumor is distinctly cystic in character and has not the ill-defined borders of an intraperitoneal hematocele, and it is usually more movable and lies higher than a broad-ligament hematoma. If we are denied the evidence of decidual membrane and amenorrhea the diagnosis is very difficult, and is based chiefly upon the pedunculated character of the tumor, the tort pedicle of which we may detect by rectal examination. In torsion of an ovarian pedicle the uterus is often drawn to the side of the tumor, while in extra-uterine gestation it is invariably displaced in the opposite direction.

Intestinal Perforation: The pain which attends the rupture of a pregnant tube may be referred to the epigastrium. I once observed this peculiarity. There was no pain or tenderness in the lower zone of the abdomen. Guided by this symptom pain alone, we might mistake tubal rupture for a gastric or intestinal perforation. The clinical history, the character of the shock, and pelvic examination should render such error unpardonable.

PROGNOSIS.—Intraperitoneal rupture in the early weeks is especially fatal from free and continuous hemorrhage. Extraperitoneal rupture is a rare course; undoubtedly many such cases recover with the destruction of the embryo. Interstitial pregnancy, with its late rupture, usually about the fourth month, is the most serious of all forms of ectopic gestation. When a hematocele is formed the mortality is not so great, Voigt reckoning it from 25 per cent. to 28 per cent. In 132 cases palliatively treated, collected by Price, the mortality was 50.3 per cent. In Winter's series of 241 cases, which were left entirely to nature, 166 died; there was hemorrhage into the general cavity in 128 instances, with 121 deaths.

Price's operative mortality is 3.61 per cent. In 326 cases collected by him, operated upon by various surgeons, including 83 of his own, the total mortality was 13.49 per cent. Martin has collected 585 cases in which operation was done, with mortality of 23.4 per cent. Boldt has operated 62 times, with 58 recoveries.

I have operated 58 times, with 6 deaths directly following operation, and 1 death eight weeks after from suppurative hepatitis.

SURGICAL INDICATIONS.—It may be very safely assumed that the only proper treatment of ectopic gestation lies in a resort to surgical measures. The time and method of procedure are questions not as yet fully settled, and require the most astute surgical judgment under alarming and trying conditions. It may be further stated that, with limited exceptions, ectopic gestation should be dealt with through celiotomy. We recognize the prowess of Martin, and fully appreciate the work of Kelly, the two leading surgeons who most strongly advocate colpotomy as a preferable procedure, preferring celiotomy in exceptional cases. Prompt abdominal section is the course pursued by those most actively engaged in this work; Prochownik considers it the only course; Dührssen advises its early application; Ségond in only a few cases resorts to colpotomy; Henrotin, though an expert in vaginal work, advocates abdominal section except in septic hematoceles.

With this unanimity in the method of procedure, it only remains to discuss the indications under the special conditions. From Parry's time to the present day this condition has been likened to a wound severing an artery. This forcible illustration has served its purpose, and everyone is now alive to the dangers of rupture. While granting that intraperitoneal hemorrhage is a condition of alarming emergency, yet it seems appropriate in the interest of both patient and surgeon to advise deliberation and to caution against submitting a moribund patient to any surgical procedure. We occasionally see reports of cases, and some of us have had the gratifying experience of rescuing from profound shock these patients with ruptured tubes. Yet, if I mistake not, if it were possible to collect the statistics, we would find that the patients dying of shock one to four hours after operation would far exceed the list of those recovering under similar conditions. The very pertinent question then arises: In a case of primary rupture with intraperitoneal hemorrhage and shock of profound degree, should we resort to immediate operation or should we await some evidence of reaction in response to aiding stimulation? Death immediately from the first shock without some reaction is exceptional; I have in this article recorded two cases. If carefully watched and duly stimulated we shall note

some increase in the volume of the pulse, some lessening in its frequency; slight color will return. This reaction may be only transient, yet it is the accepted time for interference. In any case that is continuously bleeding, the pulse will increase in frequency every quarter-hour, and this increasing frequency calls for prompt interference. My master, Mr. Bantock, confidently expected and usually waited for these evidences of reaction. Kelly would prefer to wait a few hours if there are encouraging signs of improvement. Taylor, and Cullingworth caution against operating in profound shock. In this sufficient is said to interpose an objection to the rather heedless advice to "operate immediately."

When ectopic gestation is recognized before rupture or in the condition of tubal mole without appreciable intra-abdominal leakage, we should, after suitable preparation, remove the pregnant tube. More conservative work has been done by Martin, Kelly, and others, by simply incising the tube, evacuating its contents and suturing the wound in it, or by resecting portions of the tube. Martin has employed this method in 9 cases, and all by anterior colpotomy. We doubt if this would become generally applicable. The special indication for its employment would be an absence of the opposite adnexa. In all cases submitted to operation in this early stage, tubes and ovaries of both sides should be examined. It is not my purpose to enter into the technic, but it is, perhaps, not amiss to say that in removing a pregnant tube the infundibulopelvic ligament and uterine end of the tube should be secured by separate ligatures, the tube cut, and the intervening space in the severed broad ligament sutured over. The conditions of the broad ligament and tube in ectopic pregnancy are rather favorable for infection, and the above method avoids the use of large ligatures and a massive stump.

With intraperitoneal hemorrhage from rupture or abortion, the patient being in operable condition, the abdomen should be immediately opened. The first indication is to clamp the uterine end of the Fallopian tube, preferably with Doyen's light elastic clamp, then in a similar manner the outer border of the broad ligament. An assistant should be constantly employed with hypodermoclysis. The patient should not be elevated to the Trendelenburg posture until such clots as are accessible have been

ladled out with the hand. My patients have had a better convalescence when the abdomen was moderately clean; yet no time should be consumed in attempting the removal of all free blood. With the larger clots of blood removed, we may elevate the patient's hips and proceed more leisurely to the ligation and removal of the tube; and if the condition of the patient permits, deal with the other appendage as indicated. We must bear in mind that an anemic patient should under all circumstances receive the minimum amount of anesthesia, and that this is not the occasion for any fancy work. The sooner the patient's abdomen is filled with saline solution and properly closed the better the result.

In recent cases of intraperitoneal hemorrhage drainage is not demanded, but in all old cases vascular tufts appear in the walls of the hematocele. The hemorrhage from these is controlled by hot sponge packing. If more oozing is anticipated than would be safe to leave to the peritoneum, vaginal drainage with strips of gauze is desirable.

In ruptured interstitial pregnancy the tissues of the uterus will rarely bear secure suturing, and the safer procedure is hysterectomy.

If, after exposure by abdominal section, the tubal rupture proves to be extraperitoneal, I have resorted to one of three separate procedures: (1) I have opened the broad ligament at some fluctuating point, having previously protected the general cavity with gauze pads, and with two fingers introduced have cleaned it of its contents; (2) I have found the anterior fold of the broad ligament so elevated that I have made an incision parallel with Poupart's ligament, entered the subperitoneal space, evacuated it, and packed with gauze; (3) I have followed the method of Kelly, opened and drained through the vagina without disturbing the abdominal aspect. In early pregnancy the last is certainly the best procedure.

In old-standing hematocele, showing evidences of infection with suppuration, vaginal incision and drainage is the proper measure. The vent should be free, gentleness must be used in evacuating the pockets, under no circumstances should the cavity be distended with fluid, and, as Henrotin advises, we should drain with numerous narrow strips of gauze.

The management of ectopic pregnancy with living child and quick placenta after the fourth month is a controversial question. The ectopic child is not an individual with sufficient claims to life deserving of that morbid sentimentality which holds that its and the mother's rights to life are coequal. Werder shows us that of 40 of these children delivered alive, 18 died within the first week, and only 5 lived longer than eighteen months. Orillard has collected 60 cases of operations with viable children; 27 were delivered alive, but 26 soon died; 6 of the 60 were deformed. The fetal life should be humanely regarded, but never at the sacrifice or jeopardy of the mother.

An ectopic gestation is analogous, says Werth, to a malignant tumor; hence prompt action is demanded. It is impossible that the experience of any one surgeon is sufficient for him dogmatically to outline rules for guidance; but from the combined experience of others I conclude that from the fourth to the seventh months, if an ectopic gestation is recognized, we should immediately proceed to remove it. After the seventh month, in the interests of the child, we should wait until near term. And now comes the rub. The modern teaching is to operate before spurious labor, believing that the dangers of hemorrhage are not greater than the probabilities of infection with its almost certainly fatal ending. Sutton's statistics are almost conclusive: in 16 laparotomies performed after the death of the fetus about full term, all the mothers recovered; in 13 operations performed while the child was alive, 8 mothers died and only 2 children lived. For the ten years from 1889 to 1898 inclusive, Harris collected 40 cases of operations for ectopic viable fetuses; of these 27 mothers (67.5 per cent.) recovered.

Statistics aside, we know that the placenta retains its vascularity for an indefinite time. We know that while procrastinating in the interests of the child fatal hemorrhage may occur, and that there is the ever-present danger of infection. Pozzi thus beautifully disposes of the question as to the propriety of waiting for spurious labor and the death of the child: "One may obtain no advantage from delay, which necessarily sacrifices the life of the child and exposes the mother to fresh complications. Here, as in nearly all problems of abdominal treatment, the theoretical objections of the timid disappear before the brilliant results of

courageous action with skill." It therefore appears that after the seventh month, the child known to be alive, we should watchfully wait to within two or three weeks of full term, then proceed with the undertaking, the ideal consummation of which consists in the removal of the fetus, placenta, and gestation-sac. That such complete work is rarely accomplished a study of reported cases will show.

Usually median section is selected. It may be deemed advisable, because of the recognizedly lateral position of the child, to approach through a lateral opening. The first desideratum is to remove the child, which may be found free in the abdomen or else enclosed in its gestation-sac. The next step is directed to the management of the placenta. In 1894, Cullingworth thus wrote: "We should empty the placenta of blood and close the wound hermetically upon it." This advice is supported by Olshausen, Sutton, and Taylor in some instances. Ségond says that, in general, intervention should be limited to extraction of the fetus with simple marsupialization of the sac and abandonment of the placenta. Price, also, advises to leave the placenta to come away piece-meal. This rule cannot always be observed, and it need not, for a placenta may be in some instances very easily enucleated, and in other cases the change of tension consequent upon removal of the child and evacuation of the liquor amnii may cause partial separation, demanding enucleation as a hemostatic measure. If enucleation of the sac and placenta is determined upon, ligation of the ovarian artery will in a great measure control the hemorrhage. In some instances it may be advisable to ligate the ovarian artery upon the pregnant side, then proceed upon the sound side, ligating the ovarian and uterine arteries, cutting across the uterus at its vaginal junction, ligating the uterine artery of the pregnant side, and enucleating from below. Enucleation seems to have been a very easy matter in several cases, notably in Eastman's and in Dunning's.

In tubo-abdominal pregnancy the placenta receives its nourishment largely from the Fallopian tube, and Mr. Taylor argues that wherever the placenta is there is the Fallopian tube. In such cases, after removal of the fetus, we can secure the ovarian artery at the pelvic brim and the uterine near the cornu of the uterus, thus controlling the blood-supply of the placenta, and proceed,

usually with safety, to the removal of the placenta. Mr. Taylor holds that in all cases of tubo-abdominal pregnancy the placenta should be removed; and that in tuboligamentary pregnancy there is not the same necessity for removal of the placenta. Thorough provision should be made for drainage.

It seems that the conditions are rarely present that justify an attempt at enucleation of the sac. The surgical sense of the operator must guide him in determining to attempt the removal of the placenta. After the death of the fetus the removal of the placenta is generally a simple matter. With dead fetus and suppuration the operator should be content with emptying the sac and providing drainage.

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INDEX.

ABBREVIATIONS: A. = Artery. Abdom. = abdominal. Carcin. = carcinoma. Diag., differ. = diagnosis, differential. Surg. indic. = surgical indications. Tuberc. = tuberculosis.

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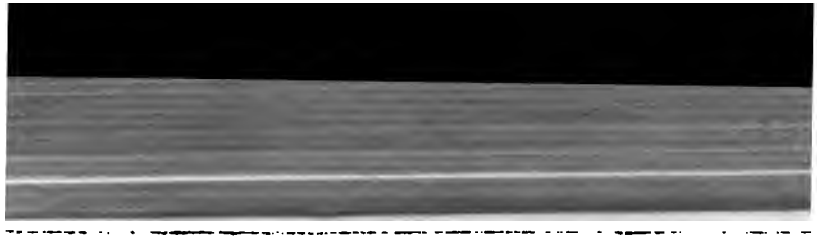

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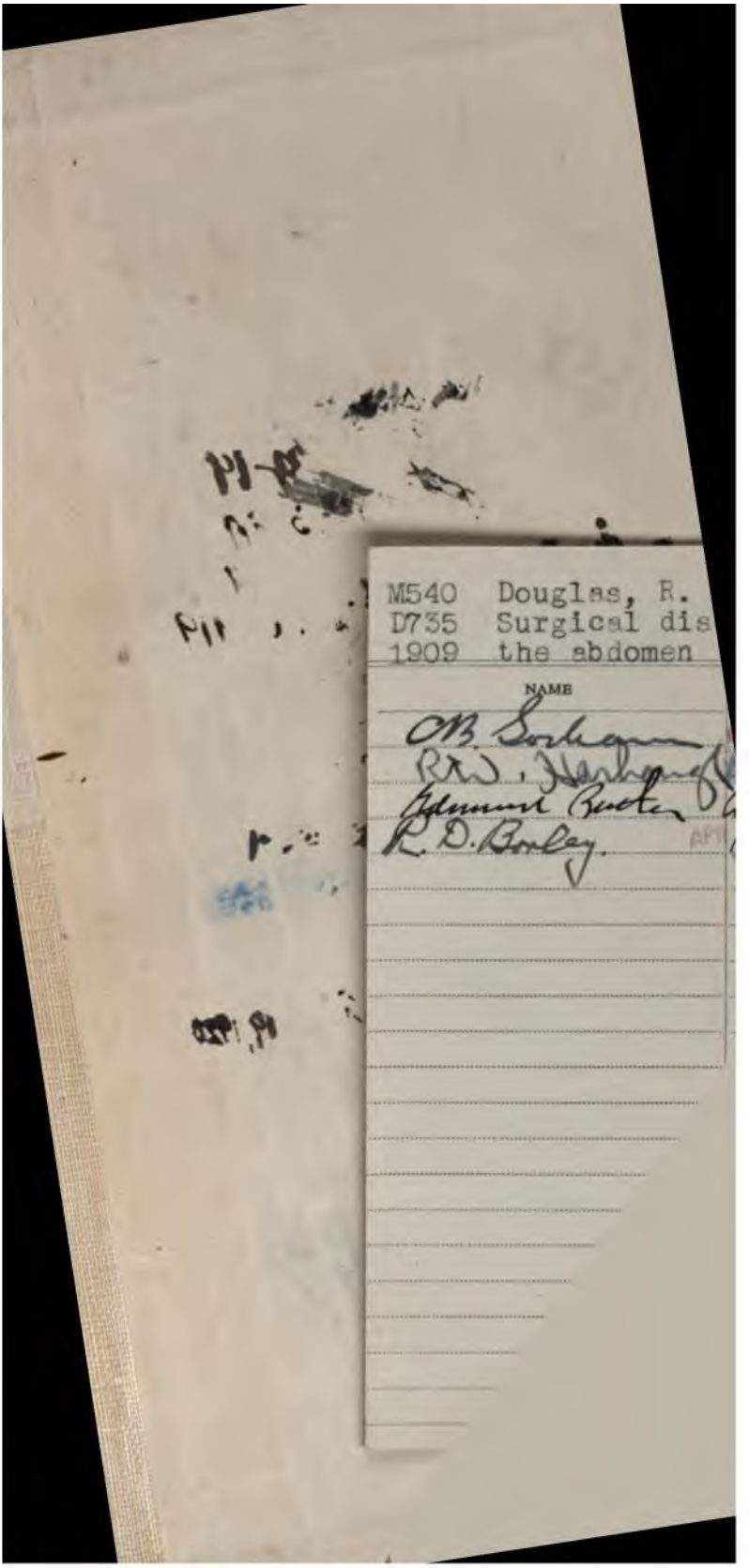
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